VOLUME XVIII NUMBER 4

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DISEASES *of the* NERVOUS SYSTEM

A Practical Journal on Psychiatry and Neurology

APRIL
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1. Livingston, S., and Petersen, D.: New England J. Med. *254*:327 (Feb. 16) 1956.
2. Pence, L. M.: Texas State J. Med. *50*:290 (May) 1954.
3. Berman, B. A.: Am. J. Psychiat. *112*:541 (Jan.) 1956.

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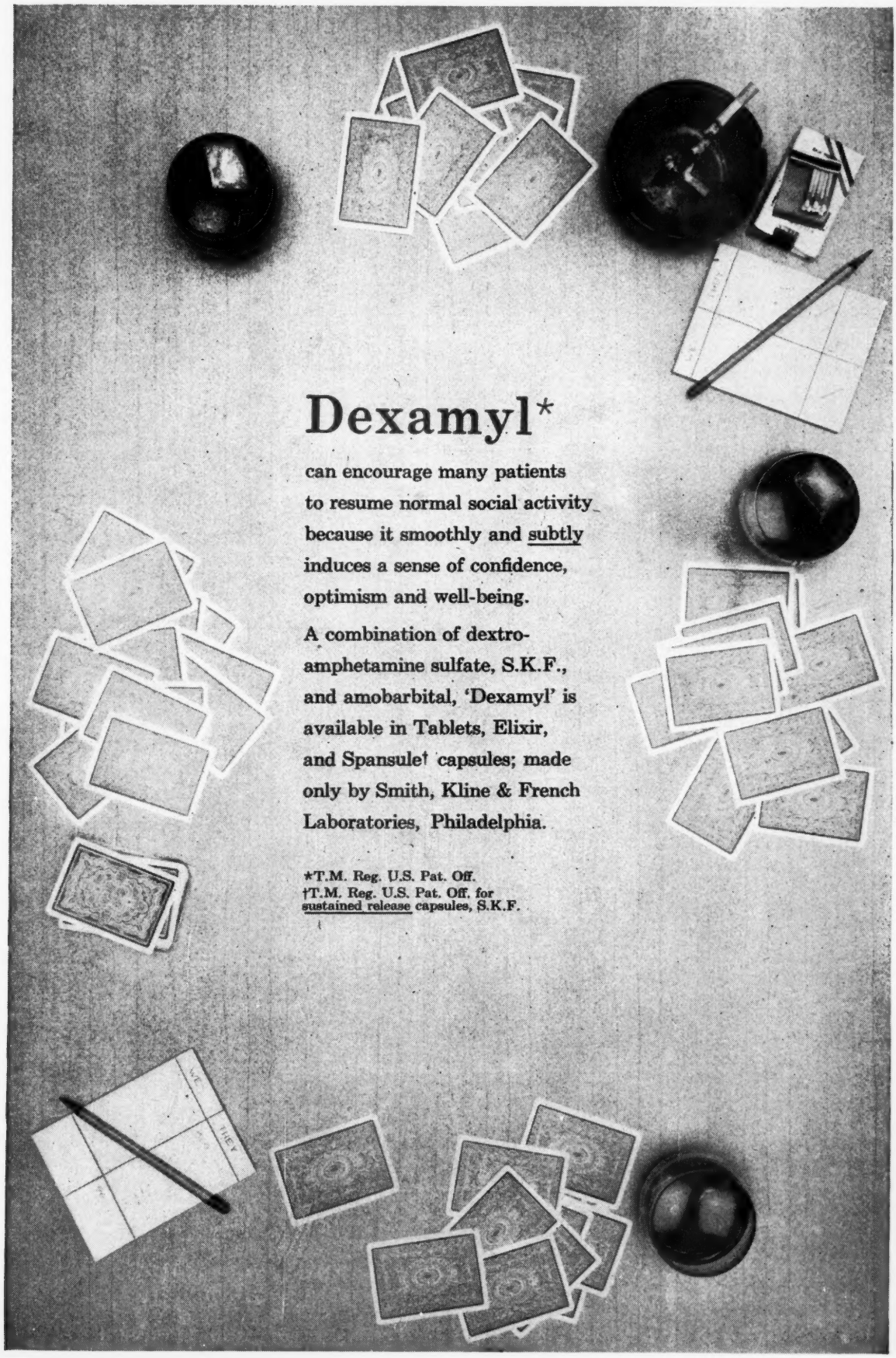
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1. Freed, H., and Peifer, C.A.: Am. J. Psychiat. *113*:22 (July) 1956.

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DISEASES of the Nervous System

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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Arteriolar Mycosis Associated With Chronic Degenerative Brain Disease

(A New Look at Sclerotic Patches)

JAMES W. PAPEZ, M.D. and B. PEARL PAPEZ

Sclerotic patches on arterioles and capillaries in the cerebral cortex of psychotic patients have been studied during the past two years under oil immersion, phase contrast microscopes. Small bits of cortex with fine vessels were placed on microslides. The pieces were covered with sterile normal saline and the cortex was broken up or teased to expose some of the small vessels. Excess of tissue was removed and the remaining fragments of brain tissue with capillaries were covered with a coverglass which was sealed to the slide with hot vaseline. Several such preparations were made in each case. The sclerotic patches on them were then studied.

The phase contrast, oil immersion microscope admits into the condenser a ring of light around an opaque disk. This ring of light is converged obliquely at a wide angle on any object placed on a microslide. The object is thus illuminated from all sides while the vertical light through its center is excluded. The side illumination gives a view of the form of the object as if it were seen in perspective. By focusing up and down the sides of the object, its upper and lower parts can be examined. The magnifying objective also contains a central opaque disk which shuts off any central light and allows the ring of light from the object to enter the eyepiece. Such structures as capillaries, arterioles, organisms, nerve cells, axons and nerve sheaths can be visualized as objects with three dimensions. The oil immersion images of the structures we describe are fairly large and many details can be seen.

Functional evidence of cortical damage was often associated with arteriosclerosis. The part which arteriosclerosis played in psychosis was recognized by the frequency with which it was added to a clinical diagnosis of mental illness, especially in middle-aged and old persons. It was in fact, a common finding at autopsy which usually referred to the condition seen on large vessels. It was also found in senile persons without psychoses. The blood pressure findings in essential hypertension tended to imply a sclerosis of small terminal vessels wherein it impeded blood flow. Abnormal deviations of blood pressure were common. In older persons there were other clinical signs, such as inability to sustain mental tasks, loss of memory, confusion, disorientation and a general impairment of intellectual functions. These were regarded as symptoms of cortical damage associated with arteriosclerosis. In fact, the walls of the vessels, and nerve cells of the cortex were involved by a common mycotic infestation and each contributed to the signs. These two conditions were not limited to adults and old persons.

Arteriosclerosis was prevalent in brains of patients with mental illness, especially in the senile group. Very few patients escaped the finer grade of sclerosis which affected the small cerebral vessels.[19] Even a careful scrutiny of the fine injected arteries on the surface of the brain failed to suggest the intensity of sclerosis of small vessels in the underlying gray matter.

Our research showed that these arterioles and capillaries were subjected to an intense

---

Demonstrated at meeting of American Neurological Association, June 18-20, 1956, Atlantic City, N. J. Read before Amer. Assoc. Physical Anthrop. April 7, 1956, Chicago, Ill.

From Department of Mental Hygiene and Correction, and Laboratory for Biological Research, Columbus State Hospital, Columbus, Ohio.

infestation by myotic spores which came from the nerve cells as well as from red blood corpuscles within the vessels. We have come to recognize a triad of related sources of mycotic infestations, namely the red blood corpuscles, nerve cells and the mycotic patches in walls of blood vessels. We wish to present the fundamental fact that the well-known sclerotic patches on small and large blood vessels had a mycotic origin in common with that seen in blood corpuscles and nerve cells. They were a manifestation of a chronic (slowly progressing) disease which imposed a severe handicap on the tissues which the vessels supplied and on the vasomotor stability of the individual.[1,2,3,4]

The mycotic patches on blood vessels were seen in young patients in the first and second decades of life associated with mental deficiency. We had 16 cases in the first decade, six in the second and a number of biopsies from schizophrenic patients in the third decade. In the first decade the patches were small, composed chiefly of dot-forms and ring-forms and small mycotic cells without fat around them. In the second decade the cells increased in size, became compressed or polyhedral, and the central colloidal substance was increased in amount, as well as the fatty (cholesterol) envelopes.[5] Together they formed a thickening or elevation on the vessels. We gained the impression that it took as long as 12 to 18 years for the mycotic cells to mature and grow reproductive filaments in their colloidal centers. It was a slow chronic process. Meanwhile new patches were forming in other locations. The mycotic spores were minute, fuzzy elementary bodies, dot-forms or older ring-forms which infested the blood stream, parasitized the red blood corpuscles, or entered the tender walls of arterioles and capillaries of these children (Figs. 1 and 2). It was these small mycotic forms which made the beginnings of sclerotic patches during the first two decades of life.

We have illustrated the formative stages of these patches from a number of children who died of pneumonia associated with an obscure form of "encephalitis." Figure 1 illustrates a piece of a vessel from a 6-year-old patient. The vessel was full of mycotic spores in the blood stream. Many were embedded in the thin wall of the vessel. This was an apparent beginning of a future mycotic patch. Figure 2 is from the brain of a child, aged 9. The small ring-form spores embedded in the wall of the blood vessel are seen on the left side. On the right side and below the bifurcation are seen older mycotic cells, each with a center or nucleus of growing colloidal material, pink in color. Their outer envelopes show a trace of fat deposited or synthesized around the colloidal centers. The mycotic cells in a 16-year-old patient were crowded together as they grew in size and stood out on the outer surface of the vessel (Fig. 3).

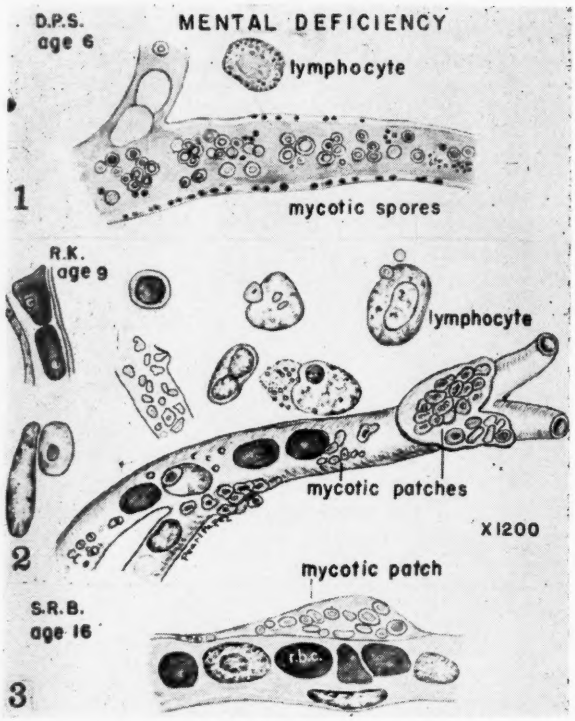

Fig. 1 shows a small blood vessel from a mentally deficient child, 6 years old. The vessel contains many mycotic spores and cells in its lumen. Small spores are embedded in its thin wall making the beginning of a mycotic patch. Above is a lymphocyte filled with spores it has ingested.

Fig. 2 shows vessels from a mentally deficient child aged 9. Mycotic patches in the vessel wall show young, growing mycotic cells. Lymphocytes above have ingested dust-like mycotic elementary bodies.

Fig. 3 shows a large mycotic patch on a blood vessel of a 16-year-old, mentally deficient patient. The dark oval structures are red blood corpuscles. Elementary bodies are seen as dust in the lymphocytes. Mycotic cells with fatty envelopes are not ingested by lymphocytes.

Brains of schizophrenics in the third and fourth decades showed well developed mycotic patches on the blood vessels in the cor-

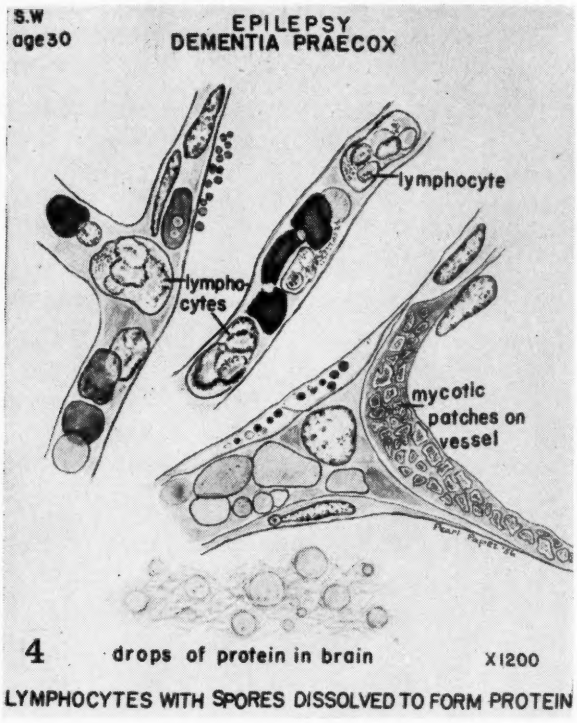

Fig. 4 shows the three stages of formation of mycotic patches in vessel walls in a 30-year-old patient with dementia praecox. In the upper vessel mycotic spores have settled on vessel wall. In lower vessel the mycotic spores have entered its upper wall and started a mycotic patch. In the crotch on the right side there is an old patch with wrinkled mycotic cells. Lymphocytes with ingested mycotic spores are undergoing lysis or dissolution. Note their broken nuclei. Red corpuscles are dark.

tex; figure 4 shows on the right side such a patch where the arteriole divides to form two capillaries. The mycotic cells are old, wrinkled and compressed into polyhedral shapes. Their colloidal contents are granular. In the upper wall of the large vessel is shown a patch more recent in origin containing large mycotic globules and ring-forms. In the capillary on the left side there is an accumulation of small globules on the outer surface of the capillary. We have estimated that it took about ten years or more[15] for the globules seen on the left side to develop into the wrinkled mycotic cells (lipofuscin granules) in the mycotic patch seen on the right side. As seen in histological sections the sclerotic patches did not give the same complete picture which we saw under the phase microscope. This was due in part to the fact that the fatty envelopes of the mycotic cells had been dissolved.[7,9,10,14]

In catatonic forms of dementia praecox it was usual to see minute, mycotic fuzzy-forms in the blood vessels. The lymphocytes illustrated in figure 4 have ingested a large number of these small spores. Such ingested spores were often seen dancing in an agitated way for several hours (6 to 24) till they underwent dissolution with the lymphocytes. These dissolved lymphocytes formed drops of mucus-like protein in the vessels[18] as well as in the brain tissue as seen in lower part of figure 4. Lymphocytes, monocytes and microglia all were the chief (res) elements of defense against mycosis.[15] To these we may add lysin.

Lysis or dissolution of nerve cells was a remarkable feature of dementia praecox (Fig. 8). It appeared to be closely associated with the drops of mucus-like protein which came from dissolved lymphocytes, broke up and wandered through the fluid spaces of the brain, till they came in contact with nerve cells. The nerve cells seemed to melt away into the surrounding spaces and carry away parts of the cell cytoplasm which contained the organisms. In this process the surface of the nerve cell and some of its dendrites sloughed off, leaving the large nucleus with only a rim of cytoplasm. We have described this cytolysis of nerve cells in dementia praecox in a number of papers.[7,8,9,15] It seemed to us that a lysin was involved in this defensive process and that some peculiarities of the schizophrenic illness might have their explanation in this cytolysis which obtunded the mind. It was inferred that the synaptic surface of the cell was damaged so that transmission of nerve impulses was blocked. The presence of mycotic auxins such as 5 hydroxytryptamine (serotonin) has been postulated as irritants at the synaptic surfaces of nerve cells in schizophrenias.[15] Abnormal growths of glia tissue were prominent findings.[19] It seemed probable that the mucus-like protein was a mucopolysaccharide[18] and a form of amyloidosis.

In the involutional psychoses of the fourth and fifth decades of life the nerve cells were severely infected with mycotic spores. Patches of mycotic cells were prominent on walls of blood vessels (Figs. 5 and 6). Much damage of cytoplasm was seen in the nerve cells. There was dissolution of cytoplasm and sloughing of its outer portions together with the organism. It was less drastic than

in dementia praecox. This appeared to be the source of the organisms that had drifted to the blood vessels and formed the mycotic patches. Histological sections showed such clusters of mycotic spores packed along the side of the vessels.[7-15] In cases that had suffered mental depression we found that the nerve cells had passed the sloughing stage and had only remains of narrow rims of cytoplasm around their nuclei. Such damaged cells had lost also some of their dendrites. In all cases there were signs of altered metabolism in the nuclei. In contrast to this, nerve cells from paranoid psychosis were usually intact with large colonies of ring-form organisms in them. In any case the presence of mycotic patches on the blood vessels indicated an earlier attack many years prior to the final one which we studied (Figs. 5 and 6).

In old patients in the 7th and 8th decades there were usually large ring-forms and large mycotic cells. We studied more than 150 brains. This suggested that the original invasion which initiated the disease had occurred long before the final illness. Two stages with a fairly long interval between them were seen in those who had passed middle age. The senile psychoses represented a severe terminal outbreak of this cerebral mycosis. Normal individuals had only a moderate mycosis. With the psychoses, the

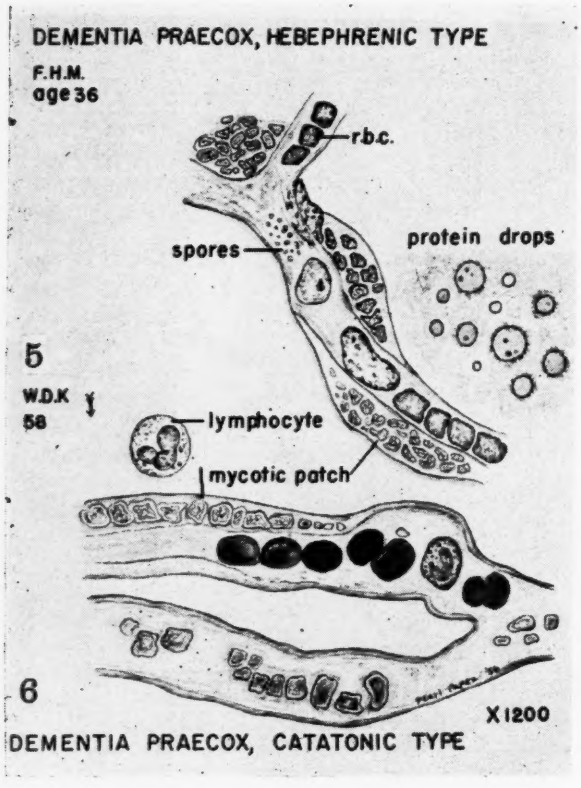

Fig. 5 shows a blood vessel with two capillary branches above in a 36-year-old patient with dementia praecox. A new patch of mycotic spores is forming below this junction. There are three old patches of wrinkled mycotic cells; one above the crotch, another to the right, and still another at lower end of vessel. Two stages of formation of sclerotic patches are thus represented. Lymphocytes have ingested the elementary bdoies and have dissolved by lysis to form protein drops.

Fig. 6 shows a vessel at bifurcation with a long mycotic patch in the wall of each vessel in a 58-year-old dementia praecox. Note the wrinkled (polyhedral) shapes of the old mycotic spores, and their thick cholesterol walls. Red blood corpuscles are dark.

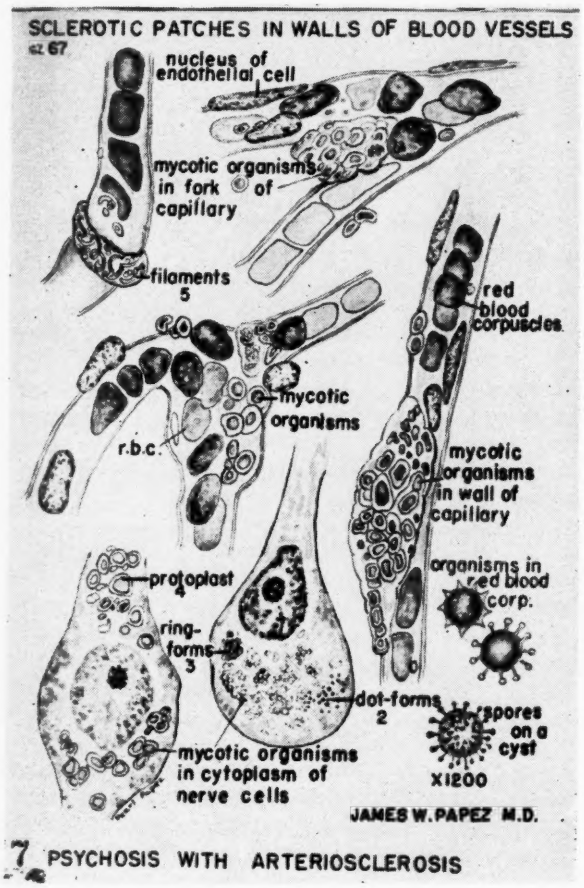

Fig. 7 shows mycotic patches on blood vessels in a 67-year-old patient diagnosed as psychosis with arteriosclerosis. Above is a drawing of a patch of mycotic cells in a crotch of a vessel. In the center is a patch below bifurcation. To the right is a patch which encroaches on lumen of vessel. Mycotic cells in their earlier stage of growth and not wrinkled, were formed late in life. Below are shown two nerve cells with old mycotic cells on left, with ring-forms in cell in the middle and with a sporulating cyst and infested red blood corpuscles on right. In upper left corner, for comparison, is a mycotic patch with filaments growing in its mycotic cells, from a 22-year-old schizophrenic.

nerve cells were severely damaged involving a greater number of the cell population. In most cases there were the large, old mycotic patches in the walls of the blood vessels, and a new crop of mycotic spores in the nerve cells. These were the two conditions usually seen in brains from senile psychoses. In many cases the clinical history referred to mental changes which began to appear years prior to the last exacerbation of mental illness.

Mycotic deposits in the brain in a case of senile psychosis are represented in figure 7. Above and on the right are drawings of three patches of mycotic cells on blood vessels. The upper one is in a crotch. Another is at a bifurcation, and a third is on the wall. Cholesterol envelopes are striking objects as seen under the phase microscope. Below are drawings of two nerve cells with organisms in them. The one to the left has, in its cytoplasm, large mycotic cells of old lineage similar to those shown in the sclerotic patches. They probably represent the first infestation in this patient. The mycotic cells are large but not numerous. The nerve cell in the middle has a multiple colony of small yellow ring-forms which may represent a second attack. Their small size is apparent. The last of the three attacks is shown on the right as a sporulating cyst which is giving off dot-forms. Above it are spore-infested red blood corpuscles. Such three successive stages were often seen in brains from senile psychoses in which a severe destruction of nerve cells took place with the last attack.

Mycosis of red blood corpuscles was present in all cases studied.[10,11,13,15] There were four viral (subfungal) strains of these spores which developed in the corpuscles. Their smaller forms infested also the plasma and bone marrow. They also penetrated the intima where they produced atherosclerotic patches in the aorta and other vessels. We have described elsewhere these elementary parasites (statoblasts) in the cardiovascular system.[11,13,15] They did not (except in one strain) produce fungal cells. They probably entered the nerve cells from the blood stream as small elementary bodies. Their neurotropic properties were apparent in young mentally deficient patients.[21]

Stages of mycotic development gave an idea of the time when each attack took place.[7,11,12,15] Time of the episodes and duration of successive attacks were judged as follows. The small mycotic cells (protoplasts) with pink or purple centers or with ring-forms were probably 6 to 10 years old (Figs. 1 to 3). The large ones with globules in them were probably 10 to 18 years old (Fig. 4). Their shrunken lipofuscin remains were probably 10 to 12 years old. The globules they produced gave origin to the next crop of spores which grew up into the wrinkled ring-forms with beginnings of central nuclei, purple cysts and globules. These might mave been 3 to 10 years old (Figs. 2, 3 and 4). Forms of recent origin in the last two years of the illness were the small, fuzzy, elementary bodies, small dot-forms on stems and ring-forms. These represented the last infectious stage or outbreak of the illness. It was suggested that the mycotic cycle was a slow one with long intervals of growth between the outbreaks of successive crops which increased in numbers with each crop.

The life cycle of these mycotic parasites was divided into two parts, one viral and the other fungal.[12] The viral or elementary body stage reproduced small spores in the red blood corpuscles. It was mostly a subfungal cycle. On the other hand, the vegetative protoplast was a true mycotic cell, the size of a red corpuscle. Within its colloidal nucleus it grew a filament that matured into globules or genomes. The globules were shed and enlarged into cysts which sporulated elementary bodies on stems as does the puffball of a fungus (Fig. 7, cyst).

Cholesterol formed the thick outer covering of mycotic cells. The cholesterol covered lipofuscin granules were the oldest residues (15 to 40 years old) of a burned-out disease with or without psychosis. The cholesterol remained, due to the fact that it could not be utilized by the body. Cholesterol when once formed, was not readily metabolized. The durable inner structure of the cholesterol molecule resisted dissolution.[5,6,10,12,14] It gave the chronic and irreversible permanence to sclerotic patches on blood vessels. Usually a shrinkage and atrophy was seen in old patches before lime deposits or scar formations took place. The

enduring nature of cholesterol containing patches in the vessels of the brain makes arteriosclerosis a formidable chronic, degenerative disease with or without mental illness.[22]

The lymphocytes and monocytes were the most frequent scavenger cells of the brain.[14,15,17] They were abundant in the blood plasma. They invaded the nervous system. They ingested the small elementary bodies and destroyed them by lysis. We assumed that they possessed a lysin or spore destroying substance. These lymphocytes with ingested spores died and dissolved into a mucus-like protein which occurred in abundance in edema of the brain, stupor, uremia and catatonic states. These drops of protein are illustrated in figures 4 and 5. They surrounded the nerve cell in figure 8. It seemed probable that the cytolysis of the nerve cells seen in dementia praecox was due to the same lysogenic property contained in the drops of mucous protein which spread to the fluid spaces in the cortex to reach the nerve cells (Fig. 8). It is known that mucopolysaccharides play a role in the pathogenesis of intimal fibrosis and atherosclerosis of the human aorta.[18]

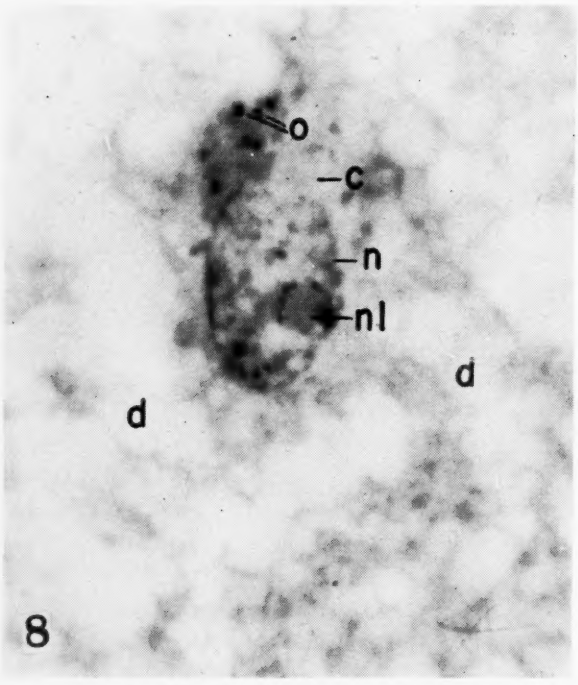

Fig. 8 is a photomicrograph of a single nerve cell from a cortical biopsy of a 22-year-old catatonic patient with dementia praecox. It shows cytolysis or dissolution of cytoplasm with mycotic spores. Cell membrane is lost and cytoplasm radiates into surrounding spaces. Note the wide circle of surrounding drops d, letter c indicates cytoplasm, o organisms, n nucleus, and nl nucleolus. This is referred to as "edema" X 1800.

Properties, affinities and pharmacological reactions of the mycotic flora have been studied. The mycotic organisms have been grown in serum broth from minute particles which passed through the finest Seitz filter, No. 3. In several weeks they grew to visible dot-forms, ring-forms and small cells with a central colloidal nucleus, when incubated at a temperature of 45° to 50° C. They were killed at 60° C. They were anaerobic, fermentative parasites. They were neurotropic for nerve cells where they lived in ATP, the Nissl substance, survived indefinitely in dead tissue, formalin and embalming fluid. They were killed by alkalies, chlorine, acetylsalicylic acid, acetophenetidin, Thorazine, reserpine and fungicides. They resembled the polyhedral viruses of insects, but grew longer filaments. They resembled the pleuropneumonia virus but did not grow on Sabouraud's agar.[16] Their relation to histoplasma was uncertain. They were found also in gingivitis, myocarditis and other chronic affections, especially those of the nervous system. They sporulated in red blood corpuscles. They grew a skein that formed globules in their vegetative mycotic cells. This two-stage life cycle was described and illustrated in reference 12.

Summary

The paper described the origin, growth and development of mycotic (sclerotic) patches on arterioles and capillaries of the cerebral cortex of mentally deficient and psychotic patients. A new method of study was employed. Teased specimens of bits of cortex were studied in saline suspensions under dark contrast, phase, oil immersion microscopes at 970 diameters. Drawings of mycotic patches from several decades of life illustrated the findings. Mycotic spores that grew in red blood corpuscles and in nerve cells were found to be specific agents which became imbedded in walls of vessels to form the patches. The spores in various stages of growth were seen in walls of vessels in mentally deficient patients. Successive steps in their development were represented in the first and second decades of life. They began

as small spores which expanded into ring-forms. These developed pink colloidal centers which enlarged to grow filaments. The filaments broke up into globules which were released and grew up into cysts. The cysts produced spores. In this process the patches increased in size and each mycotic cell was surrounded by a thick wall of cholesterol. This gave the patches the appearance of aggregates of lipofuscin granules. The granules inside the cholesterol were mycotic spores. The same process was seen in a more intense form in the later decades of life. Since growth was slow, and stages distinctive, a crude reckoning of time of successive outbreaks or crops of spores was suggested. The parts which the viral cycle in the blood corpuscles, and mycotic spores from damaged nerve cells played in arteriosclerosis were described. The production of mucus-like protein by lymphocytes in the nervous system was stated. Its relation to cytolysis of nerve cells was indicated. Properties of the mycotic flora were outlined. Cholesterol resisted absorption so that the patches remained as a serious and irreversible incubus in the cerebral vascular system.

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Value of Funkenstein Test in Predicting Psychosurgery

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A new era in psychiatry is characterized by a greater integration of the biological, physiological, social and psychological aspects of personality and disease. Such integration is part of the holistic approach. Searching for proper evaluation of a pathological condition and its therapeutic possibilities, the Funkenstein Test may prove of considerable value. Since Funkenstein and his co-workers reported that the responses of mentally ill patients to epinephrine and mecholyl can help to predict the clinical results of electric shock therapy and other modes of treatment, the predictive value of the test has been affirmed by a number of investigators. Brothers and Bennett have classified psycho-physiologic responses into five groups, based on the presence or absence of anxiety or chill, precipitated by either the epinephrine or the mecholyl or both, and seven patterns of change in systolic blood pressure have been described. So far, the classification has been primarily used as a guide in determining a favorable or unfavorable prognosis for clinical recovery following treatment with electroshock or insulin, or after psychotherapy. To our knowledge, this is the first reported case where the Funkenstein Test was used to determine the indication for psychosurgery.

The dramatic climax in the course of therapy and the remarkable results obtained, justify a detailed presentation of the case history.

B.D., a white female, age 35, was first seen (by RD) in March 1951. She had been sick for six years having been hospitalized several times and received electroshock, metrozol and insulin therapy, and psychotherapy for three years. Her complaints were sleeplessness, inability to concentrate, restlessness and irritability, many vague fears and almost constant depression. Several times she attempted suicide and had lately begun to drink. She had a "terrible temper" when drunk. Her social adjustment was poor. She lived with her mother, with whom she had repeated violent quarrels. She felt close to her brother, on whom she depended for constant service, but she was angry with him and provoked frequent quarrels because he had made her leave his home when he got married and was not always at her call. The work area was the only sphere in which she functioned relatively well. She had few dates and rather superficial relations with men. When anyone tried to come close to her she withdrew.

The following material became evident in our work with her. She was deeply impressed with the tragic aspects of her life. A sister, born when the patient was 1½ years old, had died at six months when both children were ill. The patient often thought that she should have died instead. When she was three a brother was born, and shortly after the patient started to have nightmares which were attributed to a tonsillectomy. She felt close to her father who at this time began to drink.

The parents quarrelled and her father made several attempts at suicide. He finally succeeded when she was eight years old. She was greatly disturbed and ashamed and avoided social contacts because she sensed references to the scandal. Her mother went to work and the patient and her brother were placed in a day nursery.

At school, the patient usually attached herself to one teacher whose pet she became. She wanted to please but never had much confidence in herself.

At twenty she became engaged and was married two years later. Shortly afterward she experienced her first depression attributed to a head injury sustained in an automobile accident. At the same time, however, she was having difficulties with her mother-in-law with whom the couple lived for one year.

With the exception of a few older teachers to whom she felt attached, the patient's husband was the only person with whom she had a good and close relationship. She was happy in her marriage until she learned that her husband was fatally ill, with only a short time to live, and that she could not have children. At first she took this in stride and began to work again but soon was hospitalized with a streptococcic throat infection. She became delirious and unmanageable. Then a deep depression set in and she was hospitalized and given electroshock treatment.

One and a half years later (December 1946) the patient's husband died. She went to live with her mother and brother but remained sick. She had been rather docile, gradually becoming violent and uncontrollable. Two and a half years later her brother married. She continued to live in his house but eventually felt left out and "kicked around." She had a feeling of wanting to run and her dreams revolved around insecurity which took the form of walking on the edge of an elevated track.

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The brother, under pressure from his wife and on the advice of the patient's psychiatrist, asked her to leave his home. She never forgave him for what she considered an "injustice" since she had worked to put him through school and always had taken care of him. Her temper grew worse, her drinking increased, her relationship with her psychotherapist deteriorated, and she blamed him for being put out of her brother's home. The following year, the patient had an operation for cholesteatoma in the left mastoid region. She made several attempts at suicide, each time with an overdose of pills. Finally she stopped psychotherapy and it was a few months before she consulted us.

She was cooperative and spoke freely during therapeutic sessions, but when her father was mentioned, she became upset and cried hysterically. She blamed his death for her misery in life.

An investigation of her family disclosed that the aforementioned facts regarding her brother and sister were true. She took care of her brother who was mischievous and often rebellious. She "tried to be good," was bashful and well behaved and took reprimands from her mother very seriously. She thought her mother preferred her brother and felt abandoned when her father died because he was her "friend." The mother had high standards, was critical and tried to control the family by serving and scolding them. The patient always felt her pressure. The father, in contrast, was kind and thoughtful, affectionate and easy going, had many friends and was easily pleased.

All the patient's earliest recollections were concerned with unpleasant events, the father's attempts at suicide, his death, her own fright when her mother took her to school and she refused to be left alone, her mother's order to give in to her brother, nightmares in which she would run and scream, and her fear of the dark. Her only happy recollection was of her father's joy when her brother was born, and she was happy because he was happy.

Her life style was summarized as follows: twice dethroned, then deserted by her father, never sure of her place, she tried to find it by being good and righteous but was never certain. While she rebelled against her mother's high standards, she accepted them. She tried to maintain her position by serving others and suffering through the injustices of life and of people (martyr complex). Her early recollections indicated that she did not trust life and felt horrible things were happening which she could not figure out. She considered it bad to do wrong. She felt herself a little girl caught in adverse circumstances of life and defending herself against its tragedies. Only if she was good, could she be temporarily satisfied. Her basic mistakes were the assumptions of weakness, failure to have strength, exaggeration of the need to be good and her dependence on the powers of others, whom she considered hostile in many instances.

The first year of therapy showed slow but constant progress. She was hopeless and pessimistic, but gradually gained self-confidence in the possibility of getting well. Periods of diminishing rebellion and antagonism against her family were followed by periods of increased despair, suicidal thoughts and violent outbursts, particularly in the evenings. Work adjustment improved consistently, social contacts only slightly. Her anger was transferred from her brother to her mother whom she accused of "siding with" her brother. Several times, particularly after drinking, she became violent and beat her mother. It was impossible to reduce her intake of sedatives.

In mid 1952, progress became slower and it seemed that a plateau was reached. She was given multiple courses of electroshock therapy and 120 carbon dioxide treatments with only temporary improvement. Despite all psychotherapeutic efforts her condition remained the same with disturbed periods outnumbering the periods of relief.

In October 1952, another doctor (JDF) was called in for consultation. A Watson urobilinogen test ruled out the possibility of porphyrinuria. A complete organic study indicated that the patient was organically well. With insulin therapy she showed some improvement, gained a little weight but again, the effect was only transitory. Psychotherapeutic efforts were continued. Under narcosynthesis (sodium amytal interview) she was somewhat relieved, remarking, "Why can't I always feel this way?"

During the first half of 1954 her condition worsened with increased insomnia, extreme fatigue, irritability, bitterness, and almost daily outbursts of temper. Despite the obvious psychologic disturbances of an over-ambitious, over-sensitive and socially maladjusted woman, the presence of an organic involvement was not as yet ruled out. This assumption was supported by the findings of two spinal punctures with increased cell count and positive Pandy reaction. Roentgen study of the skull and two electroencephalograms were made.

The findings of abnormally fast and slow activity indicated a "mild degree of diffused cortical injury with irritation." However, the question remained whether the positive findings in the spinal fluid and the electroencephalogram indicated an organic condition, or perhaps were the result of the electroshock treatment and an old mastoid condition.

In October 1954, the patient was referred to a neurosurgeon because of the suggestion of organic abnormality and feasibility of neurosurgery. During ten days of hospitalization, spinal fluid proved to be negative. Although the EEG still showed abnormality the patient was discharged and surgery was not advised. The recommendation was for continued psychotherapy. The patient was hospitalized for over two weeks as a second reference to neurosurgery with various psychological tests being done, including the Rorschach, Draw-A-Person, Wexler-Bellevue, TAT, and tests for generalized deterioration and frontal lobe involvement of a diffused and generalized nature although it was stated that "the organic signs may be the outcome of insulin or electroshock therapy." Pre-frontal lobotomy was

not considered as suitable treatment and the patient was returned for further psychotherapy.

Under these circumstances, further consideration of neurosurgery seemed out of the question had the Funkenstein Test not been available. The test showed a typical curve for depression, and the reaction was classified as type VI because chill occurred at the end of the procedure and after atropine was given. Such reaction is considered to indicate successful outcome for physiologic therapies.

Since previous experiments with this test had convinced us that its findings are reliable, we consulted Dr. Leo Alexander of Boston, who confirmed our view that, since the patient already had insulin, electroshock and thorazine therapy, a "graded procedure" of lobotomy should be attempted. He stated:

> "If a lobotomy of the right extent were carried out, it would certainly relieve her. It is an established fact that no type VI or VII patients remain among the patients in whom lobotomy fails. Therefore, the prognosis ultimately should be a good one."

This advice confirmed our opinion and an operation was scheduled. A bilateral transorbital leukotomy was contemplated but excessive bleeding of the right side made it advisable to postpone operation of the left side. (Surgery Nov. 26, 1954.)

Recovery was immediate and successful. The day after the operation, she received visitors who noticed a definite change in her personality. Her hands, always cold and moist, were now normally warm and without tremor.

Two weeks later the patient returned to her job. She did not manifest anxiety, her memory for both present and past events was excellent. Her insomnia instantly disappeared and she no longer required drugs. Drinking stopped entirely and she no longer lost her temper. On the contrary, she lost all hostility to her brother and mother, even making peace with the sister-in-law.

Improvement continued and one year later she was socially and emotionally well adjusted. Her insight was excellent. "A new world opened up to me I never knew before," she said. Work was the same as before, but "seemed easier," and she was able to function at higher speed with more accuracy. Her efficiency doubled and she was rewarded with a considerable increase in salary.

Her social adjustment was adequate. She went out mostly with girls, joined social clubs and enjoyed her contacts, less inhibited than before, and no longer shy. She rarely had the feeling of not belonging though this plagued her constantly before, both at work and socially.

She slept without sedatives and no longer was irritable, even during menstrual periods. Only one minor quarrel with her mother occurred and she was no longer angry with the brother. She came to the realization that living with the brother and his wife could not have worked out.

The final proof of changes in her personality makeup was the differences in early recollections. Early recollections always change with any fundamental change in a patient's personality. New ones appear, even if the same situation is remembered, it is with different details. Most dramatic is the complete forgetting of incidents remembered before treatment. In our case incidents of a pleasant nature were remembered after the operation and the frightening ones forgotten. The patient remembered how good her father had been, recalled many occasions with pleasant memories. In recalling a boat ride on the lake, she said: "I wasn't afraid—or was I?" When she spoke of her father's drunkenness and suicide she remained calm, commenting, "My hands are pretty dry, that's a good sign." Remembering his death she said actually she was not sad but felt it was all over with—the arguing and drinking. Later she missed him and even felt guilty for not feeling sad.

She did remember the unpleasant incidents which she had reported previously but recalled significant changes in the incidents. She remembered how her father asked her to read to him but did not remember that he teased her about some word though she reported this previously. One incident previously retained in memory all her life was changed significantly though it began the same as before. When she was eight she slept with her mother and the brother slept in a crib. One evening her mother decided she should sleep alone and the brother sleep with her, "because he was such a little boy." The patient was afraid to sleep alone. When she mentioned this incident she interrupted herself, saying: "No, he had diphtheria and wet his bed—that's the reason mother wanted him to sleep with her—to wake him at night when he was wet." Until this time, she always considered this incident as proof of the mother's preference for the brother. Now she realized it was not.

The difference in the patient's view of life is obvious here. She no longer felt rejected, abused or unfairly treated. She was able to accept herself and life. These observations, consistent until now, suggest not merely superficial improvement, but a total change in personality.

On May 14, 1955, a second Funkenstein test was performed. The result, compared with the earlier result, shows the relation between somatic and mental changes.

Comment

Scoville, among others, reported the results of "fractional" lobotomy in the treatment of psychoneuroses, depressions and senile emotional states without the cost to personality which more extensive procedures exact. While surgical procedure in the present case was less extensive than originally planned, the success confirms results reported by Scoville. Some interesting questions are raised:

1. Exactly what mechanism is involved in this limited procedure?

2. Had the left side been penetrated first, might the result have been different, less successful or more costly to the patient?

3. Would the effect of the neurosurgery have been the same without previous psychotherapy? The patient reports she often remembers the points discussed during her therapy and how she now can apply what she had learned.

4. Does the beneficial effect of a surgical procedure indicate an organic abnormality, or is it possible that solely psychological processes may lead to cerebral conditions which can be removed surgically?

All these questions beg an answer, despite various comments in the literature, reflecting different points of view. It can be expected that future investigations will clarify the picture.

Summary

Transorbital lobotomy brought dramatic improvement in a patient with severe depression, a patient who proved resistant to long and intensive psychotherapy combined with electroshock, insulin and carbon dioxide therapy. The surgical procedure was limited to the right side.

If the Funkenstein Test had not been available, neurosurgery would not have been performed since two leading neurosurgeons advised against the operation. The Funkenstein Test not only proved its value in predicting the outcome, but seems also to have been more reliable than all other clinical and psychological investigations to which the patient was subjected. The fact that the second test after surgery was normal, further indicates the diagnostic and prognostic value of the Funkenstein Test.

The results in this case affirms the usefulness of the Funkenstein Test in predicting favorable results for psychosurgery measures, and suggests that a unilateral lobotomy should be attempted before further surgical intervention to avoid the mental deficit accompanying more extensive operations.

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Carbon Dioxide and Nicotinic Acid for Prolonged Insulin Coma

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Prolonged coma remains the gravest danger and one of the least understood aspects of insulin coma therapy. In the course of giving insulin treatments on our psychiatric service we have had several opportunities to observe this event in varying degrees of severity. Recent work in the field of cerebral metabolism suggested to us the use of agents known to influence brain metabolism favorably in the hope of altering this post-hypoglycemic state. Two agents, carbon dioxide inhalation and large doses of nicotinic acid intravenously, were found to have ameliorating effects on the course of a stupor which was not further influenced by additional glucose and general supportive measures. In each case the response followed sufficiently promptly to indicate that these agents were of specific value in effecting the result. It was as if the metabolic processes involved were "kicked over" or received the necessary impetus to carry on spontaneously again.

In a review of the literature on this subject we found limited confirmation of the effects observed. A paper by Litteral and his co-workers,[5] described the use of a 30% carbon dioxide—70% oxygen mixture in three cases of protracted coma with good results. They postulated that a combination of carbon dioxide with pyruvate reactivated aerobic metabolism in the Krebs cycle. Cleckley,[1] described a case of protracted stupor of 20 days duration which responded to 100 mg. of nicotinic acid intravenously with clearing of disorientation and confusion in 24 hours. However, in another case large intravenous doses were given over an eight day period with no apparent effect. In this case it was noted that the facial flush usually accompanying these doses did not appear until the fourth day. Reference will be made to this situation later. It was suggested that the effect was the result of an increased demand for B vitamins concerned with carbohydrate metabolism during insulin therapy.

The etiology of protracted coma has been reviewed in an article by Spencer.[7] Although no definite generalizations can be made, he found that difficulties are more likely to occur early in treatment and with rapid induction of coma, and that cerebral damage was a function of depth and duration of hypoglycemia. Thus, a light stupor over a long period could be just as destructive as a deep coma over a shorter period. As to causation, he cited theories of cerebral vascular necrosis, anoxia, and primary failure in other organs, but favored the condition of "aglucia," a condition analogous to anoxia in which there is insufficient glucose to meet the needs of the cell. Himwich[3] also postulates a failure of glucose to enter the cell, possibly due to lack of high energy compounds required for phosphorylation. In his research on the hypoglycemic state, Himwich, found that cerebral metabolic activity, as measured by the cerebral blood flow and the arterio-venous oxygen difference, i.e., oxygen utilization, dropped to one fourth of normal in fourth stage coma. For cerebral tissue this was considered to be in the range of the lower limit compatible with life, cortical cells being the most sensitive. Reversibility in this range will depend upon the rapidity with which cellular metabolism can be restored to normal. It is our belief that general metabolic failure can be prevented when sufficient amounts of the required factors are presented in time to prevent local irreversible change.

Himwich[3] states that oxygen is present in excess of requirements as measured by the arterio-venous oxygen difference and that utilization depends on other factors. However, recent work by Lovett Doust,[6] using oximetric techniques, indicates that during insulin coma the arterial oxygen tension is profoundly reduced, from 95% to 86%, and that this drop is correlated with the onset of

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coma. Thus when tissue utilization of oxygen resumes there may be a relative oxygen deficiency. Moderate degrees of oxygen lack can be compensated for by increased amounts of glucose in cerebral metabolism, and the reverse also holds true,[3] suggesting that a synergistic effect could be obtained by increasing both substances. It has been generally noted that in hypoglycemic states the blood carbon dioxide level is moderately reduced.[3] The function of carbon dioxide in regulating cerebral blood flow is well established and, with reduction in carbon dioxide, vascular resistance increases with resulting impairment of glucose and oxygen supplies at the cellular level. Carbon dioxide-oxygen inhalations would, therefore, simultaneously open the vascular bed, increase the supply of nutrients, and lower the pH in preparation for the restoration of metabolic function. In addition an initial stimulating effect of increased carbon dioxide on nervous tissue discussed by Gelhorn,[2] may also operate favorably.

The role of thiamin, and nicotinic acid in carbohydrate metabolism is that of forming co-enzymes involved in biological oxidation. Although these elements are generally present in adequate amounts and their administration in cases of protracted coma has not always met with success, we offer a hypothesis to explain the apparent effects of large doses of nicotinic acid given rapidly by vein in our cases, with or without carbon dioxide inhalations. Nicotinic acid is known to enter into the formation of co-enzymes I and II, which function in the various schemes of carbohydrate oxidation.[3] In the Warburg-Christian Scheme e.g. co-enzyme takes up hydrogen from phosphorylated glucose in the presence of the enzyme dehydrogenase. Hydrogen is passed on in "bucket brigade" fashion through, cytochrome reductase, cytochrome-C, and finally combines with oxygen to form water. Co-enzyme is also necessary for the activation of the Kreb's Cycle by conversion of pyruvate to acetyl Co A. Thus, nicotinic acid, in readily forming large amounts of co-enzyme, may significantly assist the oxidative process by providing receptors for all available hydrogen.

Method:

Insulin coma therapy on our service is given to schizophrenic males under forty without physical abnormalities and with a normal electrocardiogram, fasting blood sugar, and skull, spine, and chest x-rays. Insulin is given at 7:30 A.M. in dosage adjusted to produce, within three hours, a one-hour coma in the third stage according to Himwich.[4] The comas are normally terminated with 100 cc's of 33⅓% glucose intravenously followed by oral feedings. With delayed awakening, this is followed by an additional 100 cc's glucose with 100 mg. of nicotinic acid intravenously. If one half hour has elapsed without progressive signs of response, a protracted coma is felt to exist. A continuous intravenous drip is then started consisting of 10% glucose in water to which B-complex and C vitamins and potassium chloride are added. At the same time nicotinic acid is injected fairly rapidly, in amounts from 200 to 600 mg and repeated at short intervals, if a favorable response is obtained, until the patient has recovered. If no response is obtained, the patient is given inhalations of 70% carbon dioxide—30% oxygen through a five liter breathing bag. After five or six breaths there is usually some response and the mask is removed to evaluate this. It may be necessary to repeat this procedure two or three times to maintain the desired state of awareness.

The following cases of prolonged coma occurred on our service over a 20 month period during which time some 70 patients were treated. Two cases were treated with nicotinic acid alone and the others were given the combined treatment.

Case I. J. A. G., age 34, had 20 hours, 30 minutes of previously uneventful coma. His 33rd treatment on March 28, 1955, was combined with electroconvulsive therapy, given after 70 minutes of third stage coma on 180 u of insulin. Termination of coma after ten minutes was attempted but no response was obtained. He was noted to have dysconjugate eye movements and positive Babinski's but the corneal reflex was present. Some improvement resulted with 150 mg. of nicotinic acid placing him in second stage coma responsive to stimuli but out of contact. One half hour later, after a total of 100 grams of glucose and 300 mg. nicotinic acid intravenously, carbon dioxide inhalations were started. After six breaths he pulled away crying,

"What the hell are you doing?", following which he lapsed back to his former state. The procedure was repeated with 200 mg. of nicotinic acid, and following this he remained in contact but was acutely anxious and screaming at intervals. Forty-five minutes after the treatment was started he seemed completely oriented but complained that he was not awake and insisted on getting up to walk the floor. Cortisone, 100 mg. was given and later in the afternoon, after a short sleep, he awoke, ate and stated he felt well. Much improvement was seen in the next two weeks, following which he again became highly obsessive.

Case II. R. L. S., age 29, had received a total 15 hours of coma uneventfully. On May 16, 1955, after 50 minutes of coma on 200 u of insulin he responded as follows: He got up and took a shower but did not speak. About two hours later he began to talk incoherently and was unable to make any intelligible remark. However, he could understand questions and was able to nod yes or no to any question asked him. When attempting to talk, a garbled succession of syllables was produced. He was able to move about and perform any task requested and seemed distraught about the fact that he was unable to communicate verbally. He was given 50 grams of glucose intravenously and 1250 mg. of nicotinic acid intravenously over a half hour period. He then began to say a few intelligible words. During the next half hour he received an additional 50 grams of glucose and 1250 mg. of nicotinic acid intravenously and at the end of this time was able to communicate verbally in normal manner. His subsequent course was uneventful.

Case III. W. R. M., age 27, had five hours of uneventful coma time. On May 31, 1955, he received 180 u of insulin following which he had 60 minutes of third stage coma. He did not respond to the usual termination procedure, remaining in a light second stage coma in which he was extremely hyperactive, combative, not responding to questioning and swearing in Spanish although this was not his native language. During the next two hours he received an additional 145 grams of glucose intravenously, plus 1500 mg. of nicotinic acid intravenously. At the end of two hours he began to respond to verbal questioning and started to take fluids by mouth. His condition rapidly improved and his recovery was uneventful.

Case IV. C. D. L., an 18 year old negro had uneventful comas for a total of eight hours and 40 minutes. On October 19, 1955 he failed to respond to the usual termination procedure after 60 minutes of third stage coma on 200 u of insulin. He fluctuated between low second stage and third stage coma. He failed to respond to an additional 150 grams of glucose intravenously and 1500 mg. of nicotinic acid intravenously and was given 70% carbon dioxide and 30% oxygen mixture approximately an hour and 15 minutes after the original termination procedure was attempted. After six inhalations, the patient immediately answered "yes" when his name was called but would say no more than that and then rveerted back to the former restless and incoherent state. After three additional brief periods of carbon dioxide inhalation, the patient was answering questions and able to take oral feedings, although he remained somewhat confused. An additional 100 mg. of glucose, 2500 mg. of nicotinic acid and 100 mg. of hydrocortisone were given intravenously followed by 250 cc's of whole blood. The patient continued to improve and within three hours following the initial termination procedure he was considered recovered.

Case V. C. E. H., age 35, had completed two hours and 20 minutes of coma time. On February 21, 1956 he received 320 u of regular insulin and following 55 minutes of third stage coma failed to respond to the usual termination procedure. In the next half hour he was given a total of 600 mg. of nicotinic acid and 150 grams of glucose intravenously without response. He was then given 70% carbon dioxide and 30% oxygen. After seven or eight inhalations he could answer "yes" when his name was called and repeated several words. The inhalations were repeated three times as the patient would tend to lapse back into a low second stage coma following each improvement from the carbon dioxide. After four series of inhalations over a period of about 15 minutes, his condition stabilized and he was then cognizant of his surroundings, able to answer questions and take oral fluids. His recovery was uneventful thereafter.

Discussion:

In the course of treating these cases we found that response was optimal when the larger doses of nicotinic acid were given, followed shortly by carbon dioxide inhalations. The merits of the 70% carbon dioxide mixture have been described in articles by LaVerne.[4] The oxygen tension in this concentration is adequate to provide maximum oxygenation of the blood while the high concentration of carbon dioxide provides a prompt alteration of the blood value which reduces the time of inhalation considerably and achieves a clearly defined change in the level of stupor. In order to confirm the general level of blood carbon dioxide we obtained samples on all patients in coma and found that the levels ranged in the lower limits of normal, between 19 and 22 volumes per cent. We found that one could not assume the blood glucose to be at adequate levels in spite of large amounts of intravenous glucose, for, in one of our cases, despite 300 grams having been given over a three-hour period, the blood sugar was reported as 60 mg.%. The level of blood glucose is known

to fluctuate and lack stable regulation during prolonged coma,[3] and this may account for the frequently conflicting results of treatment measures. The apparent dependence upon adequate glucose levels for the physiological effects of nicotinic acid was demonstrated in one patient. Nicotinic acid, 600 mg., was injected before termination with glucose and the usual facial flush accompanying this dsoage did not appear and coma was unaltered. Yet, on administering glucose ten minutes later, the flush appeared promptly. This may explain the case of Cleckley, described previously, where the flush was not observed with intravenous nicotinic acid for the first four days but appeared thereafter, as being due to inadequate amounts of glucose during that period. The flush is generally considered to be a peripheral effect, nicotinic acid having no vasodilating effect on the intracranial vascular bed, but nonetheless it seems to reflect nicotinic acid activity in carbohydrate metabolism. Prolonged coma in Case II, presented in the form of a bizarre type of aphasia and this cleared following nicotinic acid alone. Spencer[7] in his review of the literature found that this symptom occurred not uncommonly in post-hypoglycemic states and that it generally disappeared within a few hours after awakening.

We have no explanation for the prolonged coma in Case I, occurring following electroconvulsive shock. This combined procedure has been used by us extensively without difficulty and indeed it has been suggested as a possible measure to treat prolonged coma.[8]

In no case in our series did the post-hypoglycemic coma persist over two hours after the initiation of counteractive measures. We attribute this to the rapid and vigorous efforts to counteract cellular aglucia and anoxia with general measures, assisted by the catalytic action of these two agents. We would welcome further trial of this method and critical comment on the hypotheses presented.

Summary:

1. A technique employing large doses of nicotinic acid and carbon dioxide inhalations in the treatment of prolonged insulin coma is described.

2. A theory as to the possible action of these two agents in correcting the underlying defect in cerebral metabolism is presented.

3. Five cases are presented in which the rapid reversal of prolonged stupor is attributed to these agents.

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A Clinical Evaluation of Pacatal

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In the past two years the number of new chemical tranquilizers has multiplied rapidly. Many of these are being made available to the medical profession and to psychiatrists in particular before thorough clinical investigations have defined their indications, contraindications, and side effects. This places the physician in a most precarious position, since he may be asked to prescribe a drug whose potential dangers are unknown and whose therapeutic action may be very limited. For this reason there is an urgent need for objective evaluations of each new compound as quickly as possible.

In 1955, Kline and Jacob[1] reported their findings on a limited number of patients treated with Pacatal, a synthetic phenothiazine derivative. This drug was being investigated in Germany and preliminary reports from there suggested that it would be useful in the treatment of certain psychiatric disorders. On the basis of their experience in which they encountered alarming bladder atonia, Kline and Jacob remarked, "it is our opinion that this drug is not a successful ataraxic and, unless means of overcoming the side effects are provided, the drug is also too toxic for clinical administration."

Since Kline and Jacob's series of patients was small and the doses of Patacal they used relatively large, there were some investigators who felt that their evaluation of this drug was harsh. For this reason the following clinical evaluation of Pacatal is submitted.

Scope of Study

For a period of one week to 4 months Pacatal was administered orally and intramuscularly in daily doses ranging from 50 mgm. to 600 mgm. to 47 men and 45 women between the ages of 7 and 70. Diagnostically, these patients were categorized as follows: Schizophrenic reactions 35; paranoid 16; catatonic 5; hebephrenic 1; unclassified 7; schizo-affective 5; and pseudoneurotic 1. Manic-depressive reactions 6; depressed 1; hypomanic 1; and agitated depressions 4; psychoneurotic reactions 46; anxiety states 21; obsessive-compulsive 22; and mixed type 3; hyperkinetic brain-injured child 4; post-encephalitic behavior disturbance 1.

Side Effects

A. General

1. Weakness and fatigue: 14 patients complained of a sufficient degree of weakness and fatigue to necessitate the addition of an analeptic (Ritalin, Dexedrine, or Meratran) to their medication. This additional medication was sufficient to overcome the weakness and fatigue.

2. Dizziness: Although no patient had any hypotensive response to Pacatal, 5 complained of dizziness on changing position.

3. Headaches: 2 patients complained that Pacatal caused transient severe headaches during the first weeks of treatment.

4. Photosensitivity: 2 patients were photosensitive while taking Pacatal. This reaction was identical in every respect to the photosensitivity produced by chlorpromazine.

5. Motor restlessness: 6 patients on doses of Pacatal of 300 mgm. or more daily developed motor restlessness of the type produced by stimulation of the extrapyramidal system. This motor restlessness was controlled by the addition of an anti-parkinsonism drug (Cogentin) to the patient's medication.

6. Granulocytopenia: 1 woman patient developed granulocytopenia which cleared rapidly as soon as Pacatal was discontinued.

7. Dermatitis: 9 patients developed some form of dermatitis. The most common were maculopapular eruptions. In 1 patient a dermatologist advised discontinuation of Pacatal. In the other patients the dermatitis cleared even though Pacatal was continued.

8. Nasal congestion: 21 patients complained of nasal congestion. Ten of them required decongestant medication for relief.

9. Dryness of the mouth: 90 patients complained of dryness of the mouth and throat. In those patients taking more than 150 mgm. a day this was a bothersome side effect. Some patients complained of difficulty chewing and swallowing which they attribute to

the severity of the dryness of their mucous membranes.

10. Visual disturbances: 80 patients complained of blurred vision while taking Pacatal. This side effect was not related to the dosage although it was severest in those taking 300 mgm. or more daily. This was the most troublesome of the general side effects to Pacatal. It annoyed patients because they were unable to read, watch television or drive a car. Several of the patients were examined by different opthalmologists, all of whom reported that the drug had caused a toxic paralysis of accommodation.

B. Cardiovascular

No untoward cardiovascular side effects were detected in this group of patients.

C. Gastro-Intestinal

1. Improved appetite: The majority of patients reported an improved appetite while taking Pacatal. Six patients developed a polyphagia with rapid gains in weight.

2. Anorexia: This occurred in 2 patients. It was associated with weight loss and no improvement in the patient's clinical condition.

3. Diarrhea: This occurred in 2 patients. It disappeared in each instance when Pacatal was discontinued.

4. Constipation: 84 patients complained of troublesome constipation. In most instances the constipation was so obstinate that frequent laxatives were required. A small number of patients also found frequent enemas necessary.

5. Paralytic ileus: 4 patients in this series developed the clinical picture of paralytic ileus. In 1 patient emergency surgery was performed because of the severity of the patient's obstruction, his rapid dehydration from vomiting and his shock-like state. The other 3 patients' paralytic ileus responded to conservative treatment following discontinuation of Pacatal.

D. Genito-Urinary

1. Urinary frequency: 30 patients mentioned urinary frequency while taking Pacatal. In some patients this appeared to be related to increased fluid intake which the patient used to relieve the dryness of his mouth and throat. In other patients this appeared to be an endocrine response.

2. Urinary disturbances: 11 patients complained of some form of disturbance in urination. The most common complaint was difficulty in initiating urination and a feeling that the bladder was not emptied completely. In some instances the symptoms were suggestive of urinary retention, although this was not substantiated by physical examination.

3. Bladder atonia: 8 patients developed definite symptoms of bladder atonia. This was marked in 3 patients requiring catheterization and Prostigmine.

E. Psychiatric

1. Depersonalization and feelings of unreality: 6 patients complained of symptoms of depersonalization and feelings of unreality. Each of these patients had an obsessive personality make-up. In each one this side effect was troublesome and necessitated discontinuance of treatment in 2.

2. Depression: 1 patient developed all of the symptoms of a clinical agitated depression while taking Pacatal. This patient had a normal response to mecholyl on the Funkenstein test prior to treatment. When retested while depressed the patient had a prolonged hypotensive response to mecholyl.

3. Toxis psychosis: One female obsessive neurotic receiving 50 mgm. of Pacatal i.m. q.i.d. developed a toxic psychosis on the fourth treatment day. This was manifested by confusion, disorientation, illusions and visual hallucinations of small people and animals on the bedsheets, floors, and walls. All toxic symptoms cleared 48 hours after Pacatal was stopped.

F. Endocrine

1. Lactation: 6 women lactated while taking Pacatal.

2. Menstrual disturbances: 3 women noted a change in the character, onset, and duration of menstruation.

G. Miscellaneous

In this group there were 2 epileptic patients. Both of them experienced an increase in convulsive seizures while taking Pacatal.

Comment

Most of these patients were ambulatory and the majority were employed. The seri-

ousness of the side effects is illustrated by the fact that of the 92 patients treated, Pacatal had to be discontinued in 64 for the following reasons: visual disturbances 38; urinary disturbances 12; paralytic ileus 4; anxiety over anti-cholinergic side effects 4; increased seizures 2; toxic psychosis 1; granulocytopenia 1; depression 1; and dermatitis 1.

Therapeutic Results

Despite the necessity of premature discontinuation of treatment because of side effects, Pacatal appeared to be a therapeutically active compound. It was most effective in the psychoses in which acute anxiety, confusion, panic and psychomotor excitement were the predominant symptoms. However, the symptomatic improvement gained was followed by a rapid relapse as soon as the Pacatal was discontinued. As with chlorpromazine, Pacatal is least effective in treating the ambulatory psychoneurotic patients.

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Sleep Paralysis, A New Evaluation

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A review of the literature on sleep paralysis and some aspects of clinical experience with it leaves me with the impression that the family physician probably encounters this problem more often than generally realized.[1-7] As described previously, sleep paralysis is a phenomenon which occurs just prior to falling asleep or awakening from sleep. The patient is aware of his surroundings but is unable to move and frequently experiences intense anxiety. In other reports it has been said that this difficulty occurs with patients troubled concurrently with narcolepsy or cataplexy. With loss of ability to move there is often an inability to speak. Hallucinatory phenomena may be present at the same time. Terms which have been used as synonyms for sleep paralysis are: protracted psychomotor awakening, parhypnotic cataplexy, awakening cataplexy, an awakening phenomenon, dissociated awakening, and pre- and post-dormitial paralysis. The paralysis may last for seconds or minutes. Subjective impression of time passage is often much increased. The patient may be released from the paralysis by outside interference that varies from a light touch to vigorous shaking.

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Attempts to explain sleep paralysis have involved theoretical constructs which generally fall into Pavlovian or psychoanalytic categories. The former approach has centered attention on neurophysiological mechanisms with reference to the uneven spread of cortical inhibitory reactions coinciding with full conscious psychological functioning. The psychoanalytic view has inclined toward discussion of unconscious drives and conflict in relation to symptom formation. Data in a previous study suggested the apparent importance of conflict over latent homosexuality in the production of sleep paralysis.[5,6] Subsequent evaluations impressed me with the role of a broader, general issue involving a parallel between the paralysis in patients and their conflict over opposing personality trends in the direction of over-all active, aggressive functioning and inactive, passive behavior.[6]

During psychiatric treatment of the patient reported here, I found that the passivity-aggressivity conflict was involved in her various problems. The following data relates specifically to the sleep paralysis.

Case Report

The patient, a 51 year old widow, presented complaints of anxiety and depression. Information regarding the sleep paralysis was given almost incidentally during discussion of other issues. Her first

attack had occurred after she was 50. Three episodes were widely spaced within the year and the description was unchanged for each. She was asleep and suddenly felt that the mattress had moved. It was as if someone had been sitting on it and had then stood up. The patient became fully aware of her home surroundings but was unable to move her body. Her eyes were closed and she was terrified. She then experienced an hallucination of someone walking around the room. Muscle tenseness accompanied her feeling of anxiety. She had the impression that a long time was passing. She was afraid to see who this person might be. After some time the patient sat up suddenly and opened her eyes. Her fear gradually subsided. Despite her paralysis and fear, the patient had the impression that the hallucinated figure was a woman whose interest in her was kindly, helpful, and sympathetic, especially in relation to the problems the patient had experienced in her lifetime. The protective aura reminded the patient of a mother-like person and association was with her own mother who had died when the patient was two years old. This association intensified her fear.

Comment

The patient's feelings and associations regarding the hallucinated figure are essentially an individual issue. Data supplied by other patients vary in accordance with their personalities and problems. The apparent onset of a first episode when she was 50 is probably an infrequent happening although spacing of three episodes during a year is not unusual because frequency of occurrence varies markedly.

There was no history of cataplexy or narcolepsy nor were they present in the patients reported previously. It is my impression that original accounts linking these phenomena can be readily explained. Cataplexy and narcolepsy are dramatic occurrences and during careful history taking it is likely that sleep paralysis would be mentioned in response to questioning. Sleep paralysis occurring only occasionally is probably mentioned rarely by the patient as a primary complaint and seems to be referred to almost incidentally during the course of investigation of other complaints. Apparently then, it occurs far more frequently in the absence of cataplexy or narcolepsy than is realized. Because of this too, I would suspect that its occurrence is far more frequent than the literature would indicate. It is likely that the phenomenon is mentioned with moderate frequency during visits to the family physician and would be overlooked if the patient made no special point of it or if the doctor were not acquainted with the symptomatology.

Supporting evidence of infrequent reporting of sleep paralysis is the fact that following my previous papers there appeared only one paper in the American literature. This dealt with a severe case treated satisfactorily with insulin induced hypoglycemia.[1]

I am inclined to believe that if sleep paralysis is of infrequent occurrence in a patient, the approach to it should be conservative and probably one of non-interference as long as no other complaints are presented. Efficacy of any treatment measures for widely spaced episodes would be most difficult to evaluate. If the paralysis is a problem secondary to other psychological conflicts, psychotherapy is indicated. If, in the case of severe, repetitive episodes, the sleep paralysis constitutes the major complaint, treatment is warranted without question. Under such circumstances the measures may be essentially psychotherapeutic or physiological, depending upon the theoretical orientation, training, and aptitude of the physician.

Summary

This is a case report on sleep paralysis in a woman, 51 years of age. Sleep paralysis is more often a secondary than a primary complaint. Lack of familiarity with the phenomenon probably accounts for few reported cases and accounts also for my belief that its occurrence is not only more widespread than has been recognized, but that the family physician may encounter it frequently. The approach to the condition should vary from non-interference to efforts involving psychological or physiological measures, depending upon frequency of sleep paralysis episodes and the background and general experience of the physician in attendance. Although narcolepsy or cataplexy may be accompanied by sleep paralysis, it should not be expected that sleep paralysis in turn will be accompanied necessarily by narcolepsy or cataplexy.

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Parental Use of Methylphenidate (Ritalin)

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In September 1955, we presented and subsequently published data[1] on the combined use of reserpine and oral methylphenidate (M.P.D.) in the management of certain mentally defective, epileptic and/or spastic patients at the Florida Farm Colony. Results with M.P.D. used as Ritalin have continued to be highly encouraging and useful in our insitution.

More recently, the introduction of a parenteral form provided us with an opportunity of using this preparation for the more severe forms of psychophysiologic depressions (i.e. depressions which may be drug-induced) or to hasten recovery from the effect of certain anesthetic agents. The immediate central stimulating effect in overcoming drug-induced sedation, depression or anesthesia and its good margin of safety led to our decision to publish this report on the same general patient population as in our earlier report. Moreover, our encouraging results with parenteral Ritalin may help stimulate further clinical evaluation by anesthesiologists, internists and pediatricians as well as psychiatrists.

As reported originally by Meier et al.[2] (working with animals) and confirmed by clinical publications[3,4,5] other than ours, M.P.D. has clear-cut central stimulant properties with minimal effects upon heart rate.

Recently it has been reported by Maxwell et al.[6] that in anesthetized and non-anesthetized dogs M.P.D. blocks the pressor responses otherwise elicited by bilateral carotid occlusion; it also produces a prompt reduction in blood pressure which had been elevated by amphetamine or ephedrine. In contrast, these investigators found that M.P.D. potentiated the pressor responses to epinephrine, norepinephrine and naphazoline. From their findings they conclude that besides its central nervous system stimulant properties M.P.D. has certain antihypertensive actions that probably are mediated centrally.

M.P.D. has little or no effect on normal or moderately hypertensive blood pressure as well as little tendency to produce an exaggerated sense of well-being or depressive rebound. In further distinction to certain other analeptic agents, average dosages of M.P.D. rarely interfere with appetite. Meier et al.[2] also noted that in appropriate parenteral dosage M.P.D. distinctly lessens the hypnotic and anesthetic effects of Pentothal. Schneider and Holden[7] found that electroencephalographic tracings in monkeys showed typical and reproducible evidence of "alerting" following subcutaneous or intravenous injection of M.P.D. They also gave the drug to monkeys with surgically induced parkinsonian-like tremors, and although the animals demonstrated typical psychomotor stimulation, the frequency and duration of the pre-existing tremors were reduced for several hours. Plummer[8] found that parenteral M.P.D. showed a clear-cut antagonism to the sedative effects of barbiturates, chlorpromazine and reserpine in dogs, rabbits and monkeys.

In our two years' experience good tolerance for M.P.D. by patients with convulsive disorders has been noted consistently. This also has been reported by Lapinsohn.[9] Ferguson[10] observed that M.P.D. given to overcome the undue lethargy produced by high dosages of reserpine also tended to control the Parkinson-like signs of tremor and drool-

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ing. Alleviation of nasal congestion produced by reserpine has also been noted by some internists using M.P.D. From the foregoing, it is seen that experimentally and clinically M.P.D. has many useful and provocative actions either alone or with other drugs.

Selection of Patients.—The selection of patients in our series was based solely upon a clinical need for parenteral central stimulant therapy either because of severe psychophysiologic depression (drug induced) or in an effort to hasten recovery from physiologic effects of certain anesthetic drugs. The majority of patients were adolescent to early adult age, ranging in age from 2 to 50 years; both sexes are included. Basic diagnosis was incidental to this study, for reasons that will become apparent. Patients were in the following therapeutic categories:

1. Clinical shock following chlorpromazine or reserpine (34 cases).
2. Barbiturate intoxication—including epileptic medication (9 cases).
3. Marked debility or moribund state from systemic infection (18 cases).
4. Recovery from physiologic effects of rectal tribromethanol for basal anesthesia or an intravenous barbiturate (thiopental, thiamylal or pentobarbital) for surgical plane anesthesia (69 cases).

Dosage of M.P.D.—The dosage was individualized according to severity of intoxication or physiologic depression. It did not appear to be related to age or weight. Dosage ranged from 20 to 55 mg. stat, repeated within 20 minutes to four hours as required. If the parenteral route was necessary for some hours or days, such as in a severely debilitated or moribund patient, M.P.D. was administered about every four hours. As a rule, the effects of a single dose did not appear to be any more prolonged than that.

Typical Cases With Each Category

1. Clinical shock, drug-induced.

A 17-year old disturbed psychotic girl, weight 132 pounds—on chlorpromazine 300 mg. t.i.d. orally for 10 days, suddenly developed severe shock. Given M.P.D. 40 mg. I.M. stat and fully recovered within 15 minutes; no recurrence.

An 18-year old mentally retarded and disturbed boy, weight 145 pounds—on reserpine 5 mg. t.i.d. orally for 4 weeks, suddenly developed moderate shock. Given 30 mg. M.P.D. I.V. stat, with return of vital function to normal within 5 minutes; no recurrence.

2. Barbiturate intoxication.

A 14-year old brain damaged spastic and epileptic, weight 50 pounds, on phenobarbital for many months. Accidental oral overdosage of 1.0 gm. phenobarbital. Twenty mg. M.P.D. I.M. produced good response within 20 minutes and was maintained on oral Ritalin during the next 36 hours.

3. Moribund.

A nine-year old brain-damaged spastic with viral bronchopneumonia, moribund, 20 mg. Ritalin I.M., good response for a period of 6 hours. Dosage was repeated every 6 hours. As of this writing, patient had been kept alive 4 days on Ritalin (total of 360 mg.).

4. Postanesthetic recovery from barbiturates or anesthetics.

A 14-year old brain damaged, spastic boy, weighing 50 pounds (correct weight) was given tribromomethanol 70 mg/kg. to permit EEG that lasted about one hour. Given 30 mg. Ritalin I.M. and reacted fully (to his limited capacity) within 12 minutes. A clinical EEG record is appended, illustrating typical response to M.P.D. following an anesthetic dose of amobarbital.

A 19-year old mongoloid idiot boy weighing 56.9 Kg., given thiopental 500 mg. I.V. for surgical procedure requiring 15 minutes. Given Ritalin 20 mg. I.V. reacted to limit of capacity within 1½ minutes.

Comments and Conclusions

Ritalin or M.P.D. was administered intravenously or intramuscularly to 129 epileptic, mentally retarded and/or brain-damaged patients ranging in age from 2 to 50 years for (1) shock from overdosage of chlorpromazine or reserpine, (2) severe barbiturate poisoning, (3) shortening the recovery period from anesthetics (barbiturates, tribromomethanol, etc.) and (4) as a general stimulant in patients moribund from systemic disease (pneumonia, etc.).

Dosage of M.P.D. was determined principally patient response. Although considerable individualization was required, ours ranged from 20 to 55 mg. stat I.M. or I.V. and was repeated within 20 minutes to 4 hours as necessary. In no instance was there any evidence of untoward effects. All except two patients responded to the initial injection within 1½ to 15 minutes. Both failures reacted well to a second dose given within a few minutes of the first. We found that in chlorpromazine overdosage treatment response began more slowly and that a somewhat higher dosage of M.P.D. was necessary at times.

Relative to overdosage of tranquilizing

agents and barbiturates, the apparently specific central stimulation of respiration and "alerting" of consciousness with Ritalin—plus its wide safety margin—appear to warrant more intensive clinical evaluation.

As an added note, the very poor basic physical condition of our patients in this study, those associated with profound chronic brain damage, accentuates the safety of parenteral Ritalin in the various conditions mentioned above.

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Mental Health Training and Research in Florida

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Many of our members were present in Nashville in February 1954 to participate in the first conference called by the Southern Regional Education Board to discuss the needs and consider means of fulfilling the needs within our southern states in mental health training and research. At that time many of the other states were far ahead of Florida in the mental health field. Indeed, many of them still are, but it should be of interest throughout the southern region to know what has happened in our state since that time. Perhaps from this group of southern psychiatrists we may glean valuable suggestions for future action.

The original committee of three requested the governor to enlarge the committee and five professional people representing all parts of the state and various disciplines were added. The survey conducted within the state was hurried but thorough and dramatically pointed up the tremendous needs in every category of mental health personnel and in the area of research. In order to facilitate the efforts of the state to become more productive in both training and research, the committee recommended the establishment, by the state legislature, of a Council on Mental Health. Backing for such legislation was forthcoming from all citizen groups interested in mental health legislation and the state legislature in its session in the spring of 1955 enacted a bill creating a Council on Mental Health composed of eleven citizens, six professional and five lay members, set up as a rotating group with a full term of four years, members to serve without remuneration but with reimbursement for per diem and transportation expense. The Council was to consult with and recommended to the State Board of Health regarding administration of the funds appropriated in the act, the awarding of training grants, and the allotments for research projects. The Council was also "to collect and prepare data on the needs for training and research in mental health, to promote the training of mental health personnel, to encourage research in mental health, and to encourage coordination of training and research activities to avoid unnecessary duplication."

The Council was to award the scholarships and stipends and prescribe regulations governing their payment and the State Board of Health was to administer and disburse the funds. The State Department of Education was charged with the duty of issuing notes and collecting them should the recipients not fulfill their obligation to practice professionally in the state of Florida for one year for each year of grant immediately after graduation. To receive a training scholarship or stipend, in addition to character and scholarship requirements, the recipient must have resided in Florida for one year. The act included an appropriation for the 1955-57 biennium in the sum of $250,000. Only $18,000 of this was budgeted for research, $75,000 having been requested. However, we were grateful for the more generous appropriation for stipends and scholarships for training in psychiatry, psychiatric social work, clinical psychology and psychiatric nursing, totaling $39,400 for the biennium, and for $9,000 budgeted for studies and survey by the Council.

The Council held its organizational meeting in Tallahassee August 30, 1955. Since that time it has awarded a goodly proportion of the budgeted stipends and scholarships. In the bill three stipends of $3,000 per year were provided for three physicians for psychiatric residency training. The three stipends were awarded in January for a six months period and for the current fiscal year (July to July) the three stipends have been awarded in full. One of two stipends of $3,000 each for resi-

Read at meeting of Southern Psychiatric Assn., Washington, D.C., Oct. 21, 1956.

dents in child psychiatry has been awarded for the current fiscal year.

During the first fiscal year five scholarships of $1,200 were awarded to first year graduate students in social work and four scholarships of $2,000 were awarded to second year graduate social work students. For the current fiscal year nine first year scholarships and four second year scholarships have been awarded. One psychiatric social work supervisor has been employed at an annual salary of $6,600 and a training supervisor in clinical psychology has been employed at a salary of $7,000 per year. One nurse specialist for in-service training in psychiatric nursing has been employed at an annual salary of $6,200 to start work January 1, 1957. One stipend of $2,000 has been awarded for a nurse to receive training in psychiatric nursing at Duke University. Four other stipends for advance training in psychiatric nursing remain unsought.

Of the $9,000 available during the last fiscal year for research grants, $5,000 was awarded to members of the staff of Jackson Memorial Hospital in Miami for equipment to be used in carrying on investigation in neurochemistry and neurophysiology related to schizophrenia and manic-depressive states. The balance of $4,000 was awarded to an instructor in clinical medicine at the University of Miami for the purpose of studying a group of elderly patients in Jackson Memorial Hospital Geriatric Clinic and a control group to determine the value of an intensive program physiotherapists, occupational therapists, recreational workers, social workers, psychologists, and psychiatrists to rehabilitate the senile and mentally ill aged.

In order to plan wisely for future expenditure of research funds, the Council and the Florida State Board of Health requested the Southern Regional Education Board to sponsor a visit of mental health consultants to the state. This was done and in May a committee consisting of Dr. Nicholas Hobbs, Chairman, Division of Human Development and Guidance, George Peabody College; Dr. William G. Hollister, Senior Surgeon and Regional Mental Health Consultant, U.S.P.H.S.; Dr. William Malamud, Head, Department of Psychiatry and Neurology, Boston University; Dr. Morris S. Schwartz, Sociologist and Consultant for St. Elizabeth's Hospital, Washington, D.C.; and Dr. Cecil L. Wittson, Chairman, Department of Neurology and Psychiatry, University of Nebraska, made a tour of the state exploring possible facilities and areas for research. The report of this advisory group has helped clarify the ideas of the Council and enable it to plan more concretely for the future. The budget request for the next biennium will include an item of $100,000 for research. It is anticipated that, because a more carefully planned program of expneding research funds will be presented than was possible two years ago, the next legislature will appropriate the full amount requested.

In addition to expending the appropriated funds as wisely as possible, the Council has performed a more general function. Last spring Governor Leroy Collins requested the Council to view two sites proposed for the Northesat Florida Mental Hospital and make a recommendation as to the site. This was done in March, the Council making a recommendation which was accepted by the Governor and the Cabinet.

The Council, among other groups, successfully petitioned a special session of the legislature for additional funds to complete the psychiatric unit of the teaching hospital being erected as part of the new medical school at the University of Florida in Gainesville. The psychiatric floor, being the topmost, had been deleted when it was discovered that the building costs would exceed the original estimates. The additional appropriation will enable the medical school to have a well equipped psychiatric department from the outset.

In August the Council sponsored a three day group leadership workshop for nurses. The workshop centered around basic understanding of human needs and interpersonal relationships. It was attended by 36 public health nurses and four institutional nurses.

At present the Council is sponsoring a conference designed to perfect plans that can be presented to the next session of the legislature providing for a mental health program in the public schools. In Florida school health is a joint responsibility of the Department of Education and the State Board of Health. There have been divergent views on how a school mental health program might most efficiently and effectively be set up on a state wide basis without disturbing the financial balance of the total mental health program. Members of the Department of Education and personnel of the State Board of Health have met in separate sessions to iron out interdepartmental differences. Next week there will be a joint session with experienced consultants to work out a plan that will be fully acceptable to both groups as well as to the public and the legislators, to whom it will be presented at a later public meeting.

During the first year of its activity the Council has met eight times for all day sessions. In the early meetings there arose numerous matters of policy and procedure which had to be settled by a group of individuals of varied background, personality and vision. Often the sparks flew. Often the equanimity of the chairman was tested. Fortunately it never faltered. At the last meeting on September 12 in Tampa all members were struck by the atmosphere of accord. The Council is closer to a sound research program and its policies for training grants in psychiatry, social work, clinical psychology and psychiatric nursing are fairly well established. In the years ahead the Council should be able to give more time and energy to other groups related to the mental health field such as the clergy and the legal profession and to broader social influences upon mental health. In his remarks to the Council at its first meeting in Tallahassee, Governor Collins set a broad goal. The Council has taken its first steps in that direction.

Eastern Psychiatric Research Association*

Reports on Use of Sedac High Frequency Stimulator

DAVID J. IMPASTATO, M.D., *Presiding*

(The following accounts are summaries of full length reports read at the meeting of the Eastern Psychiatric Research Association.)

Treatment of Neuro-Psychiatric Disorders With Sedac in General Psychiatric Practice

FERRUCCIO A. DI CORI, M.D., AND
DAVID J. IMPASTATO, M.D.

Description of Sedac Attachment

The "Sedac" is available as an attachment to the Reiter Electrostimulator. It is housed in a small box equipped with outlets for connection to the Reiter Stimulator and to the electrodes, a neon type filtering bulb and an intensity switch with three positions—low, medium and high. The potentiometer of the stimulator is used to measure increase or decrease of the current. The machine also contains a switch which when turned on shunts the Sedac automatically and connects the patient to the stimulator. This is used only when it is desired to administer convulsions (see Dr. Buckman's paper). The bulb acts as an indicator for the intensity of the current passing to the patient. The brighter the light the more current. The Sedac is also obtainable in a complete self-contained unit.

The Sedac Neuro-Stimulator attachment serves as a filter for the convulsive currents of the electrostimulator. The convulsive current of the stimulator consists of a combination of many frequencies from very low to extremely high. The current consists of groups of pulses which occur at an average rate of about 30 per second, each group containing approximately 15 pulses and each pulse consisting of a group of high and low frequency components. When these currents pass through the Sedac all the low frequency elements are filtered out and only the very high frequency values pass through. These high frequency currents retain the original order of arrangement of the convulsive current and have a frequency of a million cycles and over.

The current, especially in the lower ranges, is painless. In practice, the electrodes are connected to the patient, the current is turned on very slowly and gradually increased until the patient indicates that it is painful. At this point, the current is lowered slightly. As the treatment proceeds, the patient's tolerance increases and more current may be administered without discomfort. A treatment session lasts about 30 minutes.

The Sedac apparatus was used to treat a group of 38 unselected private patients. The Sedac Machine utilizes a current in which only the very highest frequency components of a million cycles per second and higher are used, with the machine holding back all the lower frequency components. This machine was used to treat a group of 38 patients, 16 males and 22 females. The average age was between 30 and 40 with 10 patients between 20 and 30, 13 patients between 30 and 40 and 15 patients between 40 and 60. The cases selected all had outstanding psychotic or psychoneurotic features. There were seven schizophrenics characterized by accentuated social withdrawal, abulia, inability to keep occupied and shyness. There were 31 psychoneurotic patients of whom 19 were classified as psychoneurotic mixed (clinically they showed anxiety, social maladjustment, sexual problems of neurotic nature and somatic functional disturbances); six were classified as depressed (the symptomatology was the usual mixture of anxiety features underscoring a depressive background of benign type) and six were classified as phobic (with symptomatology of claustrophobia, cancerophobia, fear of dark, etc.).

Two patients were treated every other week; five patients were treated twice weekly and 31 once weekly. The frequency of treatment was established on the basis of diagnosis and severity of the clinical picture. During the course of the treatment 21 patients were also given Methedrine (10 mg.) and Sodium Amytal (3¾ grs.) intravenously. Furthermore, a group of 32 patients were submitted to psychotherapy while six patients were not. Psychotherapy was either extensive or moderate. Extensive meant that patients were interviewed before, during, and after treatment with Sedac; questions leading to the interpretation of their symptoms were asked. Moderate psychotherapy consisted of 10 to 15 minutes general conversation during which the patients were invited to ventilate at random. The six patients who did not receive psychotherapy were guided to the room and left under the stimulation of the Sedac apparatus without the presence of the therapist. In the group of patients receiving extensive psychotherapy 11 improved, four recovered and three were unimproved. In the group of 14 patients receiving moderate psychotherapy six improved, two recovered, and six were unimproved. In the group without psychotherapy four improved and two recovered. The description "recovered" is indicated only when a total remission of symptoms occurred and the patients regained normal insight into his problems.

The Sedac method is essentially new and still on an experimental basis, but we feel that it was useful in establishing a better transference especially in those patients in whom the underlying mechanism of resistance to psychotherapy was too strong and harmful for the proper development of understanding between psychiatrist and patient.

40 5th Avenue, New York City.

*Meeting of February 14, 1957.

The Sedac Machine in the Treatment of Mental Disorders

LEO ALEXANDER, M.D., and
MERRILL MOORE, M.D.

We use the Sedac apparatus as a "training aid" helpful in administering psychotherapy. During treatment the patient appears to relax and following the treatment the change in this direction continues. He is more in contact, more willing to listen, more willing to talk, and he does talk. Emotion, rigidly repressed before, may come to the surface; women may cry softly; later they may smile. In general, patients become more responsive and more approachable, although previously they were rigid, withdrawn and "rejecting."

In interpreting the effect that we saw, we believe that the treatment in a way restored the rôle of the patient as patient and made it possible for him to accept himself in the rôle of the patient. Prior to the treatment the patient had rejected the patient rôle in that he refused help as a patient and refused to ask for help.

Apart from these psychological features, the treatment with the Sedac, which is a form of nonconvulsive stimulation, has an important physical effect, in that it induces mild cortical excitation, in contrast to convulsive electroshock which reduces the excitability of the cerebral cortex (Alexander, L.: *J.A.M.A.*, 162: 966-969, 1950). It is partly for that reason that it facilitates psychotherapy, Pavlov already having observed that psychotherapy depends upon an excitatory state of the cortex, without which psychotherapy is not feasible (Gliedman, L. H., Gant, W. H., and Tetelbaum, H.: 112th Anniv. Mtg. A.P.A., Chicago, May 4, 1956.) We may consider therefore that nonconvulsive stimulation therapy by means of the Sedac facilitates psychotherapy on the organic level by setting up a state of cortical excitation. The Sedac may also be used as a preliminary treatment to EST in anxious patients who refuse or are fearful of of EST.

The proper application of the Sedac immediately relaxes the anxious patient. The desired effect is soon achieved and the EST can be given without further alarming the patient. For this reason it is also useful if the operator of the machine, in giving instructions to his team, will not use verbal signals which might set off an alarm response. It is preferable to communicate by signs, to be ready to sustain the shoulders and to hold the electrodes in place where manually operated. Depending on the level of the patient's anticipatory anxiety, the treatment may be extended from two to five minutes.

In a series of 20 patients treated with the standard method of applying 50 seconds of convulsive stimulation at threshold level with the Reiter RC 47-D machine followed by 70 seconds of nonconvulsive countershock stimulation, the preliminary application of the Sedac did not significantly modify the immediate postconvulsive condition of the patient. It has not been able to prevent agitation if excessive amounts of current had been given to a new patient whose convulsive threshold had not been determined, while in other patients who were treated at predetermined threshold levels no significant change was observed during the immediate postconvulsive phase. However, aversion to electroshock therapy aroused by two preliminary treatments without the Sedac was considerably reduced or abolished after one or two treatments with preliminary applications of the Sedac.

We believe that anxious aversion to electroshock therapy can be inhibited by means of the Sedac in introducing a competing stimulus incompatible with anxiety which acts as an external inhibitor of the anxious anticipation of the treatment and thus succeeds in extinguishing anxious aversion to treatment. This explanation fits in with what we know of this kind of external inhibition of conditional responses in animals (Gantt, W. H., personal communication).

Boston, Mass.

Use of Sedac in Psychoses

CHARLES BUCKMAN, M.D.

Thus far we have used the Sedac as a preinduction procedure prior to administration of electrocerebral therapies. Our intent was to remove the usual anxiety, tension and fear of the currents. The Sedac was given for approximately five to ten minutes prior to the grand mal.

Two patients were given grand mal although it was contraindicated. One patient had a fractured jaw which had been wired and convulsive therapy was not considered even with a muscle relaxant. After Sedac this patient found the current to be tolerable and free from pain. He lost the immediate fear of treatment after 10 to 15 minutes and he became quiet although prior to that he was very excited. During the first treatment he tolerated the maximum currents; he even asked for additional applications. In summary, this patient, previously disturbed, assaultive, desrtuctive and unmanageable, with the use of Sedac, the symptoms were decreased by 50 per cent.

A second case was an arthritic patient, severely depressed, and convulsive therapy could not be attempted. When the Sedac was first applied he objected violently and attempted to remove the electrodes. After a few applications he lost his objections and now willingly permits the ½ hour daily treatment. He has shown some improvement.

In another group there were five cases, two of involutional melancholia. One of these was definitely relaxed by the Sedac which was given prior to electroshock and the patient became free from depression. A second case thus far has shown mild response. A third patient diagnosed as psychopathy with severe anxiety evidenced features of relaxation. The two other cases could be described as pseudo-neurotic schizophrenia. One with severe anxiety showed little response; however, the one with a great deal of psychosomatic complaints was

more relaxed and with improvement in his bodily feelings and complaints, he became more cooperative.

Our experience in two areas on seven cases, thus far, leads us to the following conclusions:

1. That the Sedac machine is a valuable therapeutic tool in doctor-patient relationship.

2. It was excellent as a pre-induction procedure in lowering the threshold of anxiety prior to electroshock convulsive therapy when combined with intensive suggestion which should lead to a reduction or elimination of drugs.

Its greatest possibilities are in the realm of facilitative psychotherapy and along the lines described by Wilcox in his use of carbon dioxide or electrostimulation for the same purpose.

Our results with this new therapeutic tool have been gratifying.

Kings Park, N. Y.

High Frequency for the Spastic Mental Defective

HAROLD H. BERMAN, M.D., and
JOSEPH SPIELMAN, M.D.

We have used the Sedac:

1. As a sedative prior to the administration of convulsive and non-convulsive therapy.
2. For the treatment of emotional concomitants and behavior disorders in epileptics.
3. For the spastic mentally defective.

This last form of therapy was suggested when a 32 year old woman while being sedated with the Reiter Sedac stated that she felt relaxed. A 37 year old congenital spastic functioning on an imbecile level was the first to be treated. His usual position was sitting in a chair with his legs crossed in scissors-spasm. The limbs could not be uncrossed either actively or passivley. After the first treatment, he opened his fingers but they soon closed. With further treatment, he voluntarily opened and closed his fingers, and has done so since. The legs did not respond. One electrode was, therefore, applied to the temple and the other to the gluteal area, and then moved up and down the leg. Almost immediately, he uncrossed his legs and flexed each separately. This he has continued to do. A second patient was a 20 year old spastic, functioning at an idiot level, very similar to the first, who developed the ability to move his limbs voluntarily. He appreciates this and "shows off" to visitors on his ward. Another patient, 32 years of age, with spasticity and athetosis, has shown a marked decrease in the athetosis, his speech became less jerky, and his ability to move in a smooth coordinated fashion has increased. A three year old child with spastic quadriplegia has been given the treatment, and has shown marked relaxation with ability to respond to her environment. A 14 year old boy, functioning at an idiot level, with congenital paraplegia, who held his arms tightly to his side, after one treatment was able to extend his arms as far as the contracted tendons would permit.

It would appear that the application of high frequency currents in some way alters the spasticity of the patients. These patients had been classified as imbeciles and idiots. Following the treatment, they were in better contact with their environment. The exact mechanisms involved are not known and must be studied. Whether neurons are still alive and capable of being revived and exploited; whether other centers can be trained to take the place of the damaged cortex or whether improvement is due to a purely physical ability making it possible to obtain better contact with the environment. Much further work must be done. We at Willowbrook are embarking on a greatly enlarged and intensive program, either to corroborate or disprove the limited findings to date.

Willowbrook State School, Staten Island, N. Y.

Intracranial Electrotherapy: Observations With the Sedac Unit

JUAN NEGRIN, M.D.

During the last few years a great deal of work has been done to develop a method to treat mental illness with an electric current sent directly to the brain through intracranially placed electrodes. This technique is based on the assumption that an electric current may give better therapeutic results if received directly by the specific brain areas than if it reaches the brain in a diffuse manner as is generally supposed to happen with "external electrotherapy." The improvement of mental syndromes and symptoms with this procedure, when external electrotherapy failed, was strong evidence in favor of this hypothesis. Satisfactory results have also been obtained in cases of intractable pain. Confirmatory data has been provided by several groups working independently.

So far, intracranial electrotherapy has been used in mental conditions only when the patient failed to respond to an intensive course of external electrotherapy, alone or combined with insulin shock. Lately, the ataraxic drugs have also been given a thorough trial. In several instances, psychosurgical operations, usually leucotomies, had failed prior to intracranial electrotherapy.

In the early stages of our work we found that results varied not only with the position of the electrodes but also with the parameters of electric current used. We have had experience with the 60 cycle sine wave, different types of uni-directional square waves and uni-directional and biphasic sharp frequencies. We have used unipolar, bipolar intracranial and intracranial combined with "external" electrodes in the outer surface of the scalp, wrist or ankle. When Reiter's new Sedac unit became available it was thought advisable to try its therapeutic possibilities for intracranial electrotherapy. We selected two patients who were chronic, deteriorated schizophrenics of long standing on whom electrotherapy, insulin shock, ataraxic drugs and psychosurgery had failed. One patient was treated last year. The other one was opreated on a few

weeks ago. It is not possible to give any adequate report on the results obtained because of the limited number of patients and the short period of observation. However, we can say that the first patient improved during and immediately after treatment to a degree that could not be obtained with the previous electrotherapies and psychosurgical techniques. Months later he had a remission but not as severe as the one following the lobotomy. We noticed that while the electric current was on, these two patients experienced no pain or uncomfortable feeling of the type occasionally present with other types of current when the electrode is placed in certain intracranial structures.

Electroencephalograms were taken before, during and after each treatment with the Sedac unit. Some of the findings were confirmatory and similar to the ones previously reported with other types of current. Other recordings appeared to be specific for the Sedac unit. A detailed report on these electroencephalographic observations with correlation of possible clinical implications will be the subject of a future report.

108 East 81st St., New York City.

Sedac Therapy in Hospital Practice

JAMES E. MILLIGAN, M.D.

Sedac therapy has been in use in our hospital for approximately eight months. We are impressed with the results. In general, the most effective application has been in treating manifestations of anxiety. Relief of distressing symptoms has been achieved in patients with both neuroses and psychoses. We are satisfied that it has a real place in therapy and expect many developments in the future.

Six representative case summaries are presented in which Sedac plays a prominent part in the treatment program. The following is a summary of these cases:

Two patients (Cases 1 & 2) had completed a course of electro convulsive treatments with remission of their psychotic symptoms. Their hospital time was being prolonged because of marked anxiety, manifested by fear, nervousness, and insomnia. Their condition improved rapidly with Sedac therapy to the extent that they are now making good adjustments at home.

Sedac was used to control many symptoms of intense anxiety in another patient (Case No. 3) so that psychotherapy could be initiated and carried on.

Following a course of CO_2 therapy, one patient (Case No. 4) still had many troublesome symptoms, including an extreme dependence on sedation. With Sedac these symptoms rapidly cleared up and enabled the man to make a good home adjustment.

Daily Sedac was considered helpful in giving a very sick and miserable patient (Case No. 5) relief and comfort notwithstanding the exacerbation of a schizophrenic reaction.

Case No. 6 is a somewhat different use of Sedac. This patient during a prolonged coma in insulin therapy became very hypersensitive, overactive, and noisy. Twenty minutes of Sedac stimulation helped considerably in controlling this situation.

Generally speaking, Sedac has been found valuable in the treatment of patients in whom such symptoms as anxiety, fearfulness, anorexia, insomnia, and tension are prominent. Relief was rapid in the cases we have followed, and appears to be lasting. We have used Sedac as the principal therapy; and in conjunction with drug, somatic and psychotherapy with good results. There have been no complications or contraindications noted.

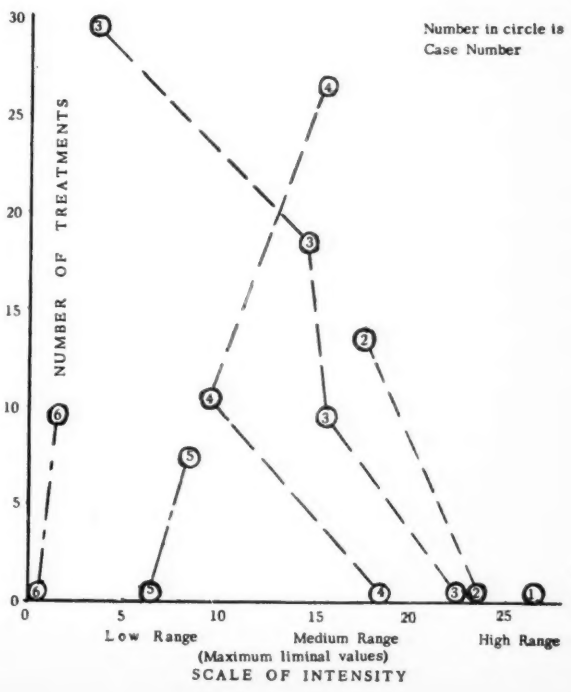
Warren State Hospital, Warren, Pa.

Employment of the Sedac for Testing and Treating Organic Disorder

ALFRED E. EYRES, M.D., AND
JACK F. WARNER, M.D.

The work presented in this paper is intended to help fulfill the need of measuring central nervous system physiology. Altschule at Harvard Medical School has measured organic dysfunction in mental illness through biochemical determinations. The Funkenstein method of testing is based upon reactivity to diametrically dissimilar drugs.

Sedac measurements on average patients indicate that tolerance to the current increases as treatment continues and symptoms diminish. However



we have gone further and found that some extreme organic disorders may respond to the Sedac treatment and that this improvement may be made in terms of exact measurement. The Sedac measures dysfunction in terms of hyposensitivity and hypersensitivity. As therapy progresses and improvement takes place the hyposensitivity and hypersensitivity measurements shift in the direction of the average normal range.

One patient (Case 3) measured a high degree of hyposensitivity. After treatment was well under way his measurement shifted to the normal range. Case 4 likewise hyposensitive shifted toward the normal after ten treatments only to later become more hyposensitive. Patient number one was measured and treated only once. He tolerated the entire maximum current output indicating severe dysfunction. This patient expired five days later. Patient number six exhibited an extreme degree of hypersensitivity which lessened slightly after nine treatments but she did not measure into the average normal range.

The flexibility of this measuring technique bids fair to become valuable. We should further investigate and make use of these measuring nad treatment techniques in psychiatric and neurological disorder.

Detroit, Michigan.

Neurological Treatment With the Sedac

SAUL I. HELLER, M.D.

Use of an electrical current in the treatment of traumatic injury to nerve and muscle tissue is an old art. This paper describes an advance in this direction; namely, development of improved currents allowing more stimulating effect with less pain than ever before possible. Patients are treated more effectively, more quickly, and more successfully even in those difficult cases which might formerly have been rejected for treatment of this nature. Electric current for these treatments is derived from the Reiter Electrostimulator filtered through the Sedac neurostimulation attachment. Effectiveness of treatment is greatly increased, so that many cases can now be treated rapidly with early improvement and without complications. Patients are more cooperative and easier to handle as a result of the reassurance provided by the early improvement.

The Sedac treatment was first utilized by me on a young man of about 27 with complete paralysis on the left side of his face. He responded immediately at the first treatment session and was markedly improved at the end of a week within the first three treatments. He was discharged at the end of a few weeks. He received a total of eight treatments.

Our next case was a young woman suffering from acute polio whose finger muscles had become paralyzed and who feared that she would no longer be able to work as a typist. The Sedac stimulation applied locally to the hand for several minutes each day gave improvement and within 10 days her recovery was complete. Frequent stimulation was possible without any side effects.

A third case was a ballet dancer who had developed an injury to the anterior serratus muscle, affecting the long thoracic nerve. There was marked residual deficit and atrophy. After eight treatments in eight days, the patient was able to state she had very little hope at first, but was able to notice improvement after the second treatment, and was cognizant of very marked improvement after the eighth treatment when she was able to lift her arm above her head. Maximal contraction of the serratus anterior muscle is obtained by stimulation deep in the axillary fossa where there is hypersensitivity to pain. Other electro-stimulatory apparatus was not tolerated.

The fourth case was a woman over 50 years of age, suffering from post herpetic ganglionitis of the sensory ganglia and motor roots of the lower lumbar area. The right leg was involved with herpes around the knees and thigh. The patient exhibited motor paralysis and sensory deficit in the herpetic lower extremity and could walk only with the aid of crutches. There was a 50 per cent estimated deficit at the start of treatment, which had been of six months' duration. After seven Sedac treatments within seven days, she was sufficiently recovered to be able to walk about the office without crutches. She has had a total of 25 treatments in a 35 day period and the estimate of recovery is at least 95 per cent. She has no need for aids in walking. Her case is of particular significance because competent authorities expressed the opinion that additional electrical stimulation would not be of benefit and considered it contraindicated.

Our results with Sedac have been excellent and have clearly indicated that stimulation with the Sedac is permissible in most, if not all, neurological disorders involving neurogenic lesions. At present, we can not conceive of any contraindications. The stimulation of effect is unusually great, and the pain is unusually small by comparison. Initial improvement is striking and the patient responds immediately. Our range of treatment possibilities has been considerably enlarged by this new development.

11 E. 68th St., New York City.

Book Reviews

SOME COMMON PSYCHOSOMATIC MANIFESTATIONS: By J. Barrie Murray, M.A., M.D., Diagnostic Physician, Tavistock Clinic Physician, Bolingbroke Hospital, London. Cloth. Pp. 285. Second Edition. Oxford University Press, 114 Fifth Avenue, New York, N. Y.

The author emphasizes the importance of recognizing the psychiatric nature of the patient's complaint either as an entirely psychosomatic, or a somatic situation with a neurotic overlay. He further stresses the importance of thorough clinical and laboratory investigation in order to assess whether the case is entirely psychiatric or is somatic with an added psychiatric element. Particularly is the clinical examination the more important because the latter when productive of negative findings frequently suggests or confirms the presence of somatic illness per se.

The physical expressions of emotional manifestations considered by the author include such syndromic pictures, their etiological considerations and exemplification of historic case material as are referable to psychogenic heart-pain and its concomitants reflected in fainting, headaches, tremulousness, fatigue and influences upon bloodpressure and pulse-rate; psychophysiological manifestations in the skin; the motor system, the commonplace low-back syndrome with its associated symptoms, as well as its differential diagnosis and its methodology of treatment when the physical basis therefor is etiologically eliminated; the various neurotic distortions and perversions of appetite and nutrition and the psychic disturbances and their varied pathological impingements upon the alimentary system ranging from globus hystericus to rectal spasm, are detailed in narrative form and the diagnosis and treatment of these visceral neuroses is discussed. The book concludes with a chapter embracing the overall topic of the psychiatric aspect of all physical disease. The book is compact, is clearly written in easily understandable language and may be used with profit by both the general medical practitioner and the psychotherapist who values the technique of clinical examination and the importance of comparison of symptoms from a specialized point of view.

Lewis J. Siegal, M.D., LL.B.

MENTAL HEALTH PLANNING FOR SOCIAL ACTION by George S. Stevenson, M.D., Sc.D. McGraw Hill, New York. 358 pp. $6.50.

In this volume a distinguished psychiatrist, who has been associated with the mental hygiene movement in this country for a quarter of a century, presents a blue print to aid us in making the most of our knowledge and facilities for promoting better mental health.

His offering consists of five parts, beginning with basic considerations, then to a survey of the various facets of mental illness. Part three concerns the protection of mental health and four, the means of elevating the subject in the minds of the people, while five surveys special considerations. All of it taken together constitutes what can be called a definitive work on the subject.

With a quiet, scientific approach born of wide experience, the author examines the varied aspects of the care of the mentally ill and the mentally deficient and finds them wanting in many respects. He then prescribes for some of the social problems involved, prescriptions which everyone connected with the field of mental health should read and consider carefully. As Doctor Stevenson puts it:

> "A person concerned with the advancement of mental health does well to select activities of top priority to absorb a large portion of his effort and resources. But it helps him maintain a balance if a portion of time and effort is reserved to cover any other aspect of the field, to seize exceptional opportunities and to deal with emergencies. The same is true of an agency such as a mental health association."

He then details the principles of program planning.

In a work like this it is difficult to pick out salient points for review, because so many of the points are salient. Many things are covered in their relation to the major problems of mental disease and health. The hospitals, public and private, the home, the school, the church, the military, all of them are considered. It is safe to say that no mental hygiene association and, in fact, no individual connected with the movement should miss this book. No matter what their relationship to the problem, it will offer them a great deal. For the physician, the psychiatrist and the student it will also be rewarding. Its breadth and its calm objective approach will broaden their knowledge and locate this difficult problem in space.

Francis J. Braceland, M.D.

THE NEROLOGIC AND PSYCHIATRIC ASPECTS OF THE DISORDERS OF AGING: Edited by Joseph Earle Moore, M.D., H. Houston Merritt, M.D., Rolle J. Masselink, M.D. Cloth. Pp. 307. $8.50. The Williams and Wilkins Company, Baltimore 2, Maryland, 1956.

The book embraces a complete recordation of the thirty-fifth annual transactions of the broad general topic and title which bears the name of the volume.

Their individually selected categories are discussed from the point of view of the differences between the neuropsychiatric problems of the aged and the more fundamental problems of aging. The scope of examination of the subject is geared toward investigative stimulation, in the sense of a dynamic rather than a static approach, with interest not so much in the broken down end-product of senility with its attendant degeneration, but is rather

directed toward a scientific understanding of how the specific wreckage of the aged individual had come about and management directed at potential preventive methods. Included in the topics considered in the volume are such categories as brain metabolism in relation to aging; the psychopathology of aging; the genetics of aging; structural alterations with aging in the nervous system; vascular disease in the brain of the elderly; neurologic changes in the aged and rehabilitation of the elderly neurologic patient. The book is generously illustrated in both black and white and color photography and each of the topics is highlighted by the individual contributor's presentation as well as the discussions which follow.

No further effort at review of the various subheads will be attempted because of the diversity of scientific treatment and approach to each presentation.

The subject matter chosen for the thirty-fifth annual session is challenging and presents a broad and interdisciplinary scientific approach to the present knowledge of the aging process, its selectivity, severity, onset and types of structural neuronal alteration encountered each of the topics presented is well documented by authoritative references and the material presented scientifically covers the subject of our present day knowledge of the neurologic and phychiatric aspects of the disorders of aging.

Lewis J. Siegal, M.D., LL.B.

BOOKS RECEIVED

MENTAL DEFICIENCY: By R. F. Tredgold, M.D., D.P.M. Medical School of University College Hospital, London and K. Soddy, M.D., D.P.M., Medical School of University College Hospital, London. Cloth. Pp. 480. $8.50. Ninth Edition. Williams & Wilkins Company, Baltimore 2, Maryland, 1956.

SUBARACHNOID HÆMORRHAGE: By John N. Walton, M.D., M.R.C.P. Cloth. Pp. 350. $7.00. Williams & Wilkins Co, Baltimore 2, Maryland, 1956.

THEORIES OF PERSONALITY: By Calvin S. Hall, Prof. of Psychology, Western Reserve University and Gardner Lindzey, Prof. of Psychology, Syracuse Pniversity. Cloth. Pp. 572. $6.50. John Wiley & Sons, Inc., 440 Fourth Avenue, N. Y., 1957.

DISEASES OF THE NERVOUS SYSTEM: By Sir Russell Brain, Bt., D.M., P.R.C.P. Cloth. Pp. 996. $11.50. Fifth Edition. Oxford University Press, 114 Fifth Avenue, New York, N. Y., 1956.

TEXT BOOK OF PSYCHIATRY: By Sir David Henderson, M.D., F.R.F.P.S., Edinburgh University and R. D. Gillespie, M.D., Cloth. Pp. 746. $10.00. Eighth Edition. Oxford University Press, 114 Fifth Avenue, New York, N. Y., 1956.

MANUAL OF ANATOMY: Edited by James Couper Brash, M.C., M.A., M.D., University of Edinburgh. Cloth. Pp. 513. Eleventh Edition. $4.75. Oxford University Press, 114 Fifth Avenue, New York, N. Y.

PRIMARY PSYCHIATRIC SYNDROMES: By Dwight L. Moody, L.R.C.P., L.R.C.S. (Edin.), Formerly Deputy Medical Supt. Springfield Hospital, Crumpsall, Manchester. Cloth. $7.50. Pp. 356. The Williams & Wilkins Co., Baltimore 2, Maryland. 1956.

EPILEPTIC SEIZURES: By John R. Green, M.D. and Harry F. Steelman, M.D. Cloth. $5.00. Pp. 165. The Williams & Wilkins Co., Baltimore 2, Maryland, 1956.

CLINICAL EXAMINATIONS IN NEUROLOGY: By Members of the Sections of Neurology and Section of Physiology, Mayo Clinic and Mayo Foundation for Medical Education and Research, Graduate School, University of Minnesota, Rochester, Minn. Cloth. $7.50. Pp. 370 with 76 Illustrations. W. B. Saunders Company, West Washington Square, Phila. 5, Penna.

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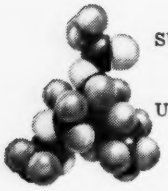

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